National Imaging Associates, Inc.*	
Clinical guidelines **BRAIN (HEAD) MRI** **BRAIN (HEAD) MRI with IAC (Internal Auditory Canal)**	**Original Date: September 1997**
CPT Codes: **70551, 70552, 70553, +0698T – Brain MRI** **70540, 70542, 70543, +0698T – IAC**	**Last Revised Date: April May 20221**
Guideline Number: NIA_CG_001	**Implementation Date: January 20232**

INDICATIONS FOR BRAIN MRI

Brain MR/MRA are not approvable simultaneously unless they meet the criteria described below in the Indications for **Brain MR/Brain MRA** combination studies section. **If there is a combination request* for an overlapping body part, either requested at the same time or sequentially (within the past 3 months) the results of the prior study should be:**

- **Inconclusive or show a need for additional or follow up imaging evaluation OR**
- **The office notes should clearly document an indication why overlapping imaging is needed and how it will change management for the patient.**

(*Unless approvable in the combination section as noted in the guidelines)

For evaluation of headache[1-5]

~~(ACR, 2019c; Holle, 2013; Quinones-Hinojosa, 2003; Schafer, 2007; Wilbrink, 2009)~~

- Chronic headache with a change in character/pattern (e.g., more frequent, increased severity, or duration)
- Cluster headaches or other trigeminal-autonomic cephalgias, i.e., paroxysmal hemicrania, hemicrania continua, short-lasting unilateral neuralgiform headache attacks (SUNCT/SUNA) imaging is indicated once to eliminate secondary causes[6] ~~(IHS, 2018)~~
- ~~New a~~Acute headache, sudden onset:
  - With a personal or family history (brother, sister, parent, or child) of brain aneurysm or AVM (arteriovenous malformation) OR
  - < 48 hours of "worst headache in my life" or "thunderclap" headache.
    - Note: The duration of a thunderclap type headache lasts more than 5 minutes. Sudden onset new headache reaching maximum intensity within 2-3 minutes.
  - Prior history of stroke or intracranial bleed
  - Known coagulopathy or on anticoagulation

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- New onset of headache with any of the following[1, 7, 8]: ~~(ACR, 2019c; Micieli, 2020; Mitsikostas, 2016):~~
  - Acute, new, or fluctuating neurologic deficits, such as sensory deficits, limb weakness, **abnormal reflexes,** speech difficulties, visual loss*, lack of coordination, or mental status changes or with signs of increased intracranial pressure (papilledema). (~~* Not explained by underlying ocular diagnosis, glaucoma, or macular degeneration~~**See background)**
  - History of cancer or significantly immunocompromised
  - Fever
  - Subacute head trauma
  - Pregnancy or puerperium[9, 10] ~~(Hamilton, 2020; Shobeiri, 2019)~~
  - Age ≥ 50
  - Severe unilateral headache with radiation to or from the neck, associated with suspicion of carotid or vertebral artery dissection
  - Related to activity or event (sexual activity, exertion, position), ~~(~~new or progressively worsening~~)~~
  - Persistent or progressively worsening during a course of physician-directed treatment[1, 11, 12] ~~(ACR, 2019c; Kuruvilla, 2015; Martin, 2011)~~

  Note: Neuroimaging warranted for atypical/complex migraine aura, but not for a typical migraine aura (see **background**)
- Special considerations in the pediatric population with persistent headache[13] ~~(Trofimova, 2018)~~:
  - Occipital location
  - Age < 6 years
  - Symptoms indicative of increased intracranial pressure, such as recurring headaches after waking with or without associated nausea/vomiting
  - Documented absence of family history of headache
  - Severe headache in a child with an underlying disease that predisposes to intracranial pathology (e.g., immune deficiency, sickle cell disease, neurofibromatosis, history of neoplasm, coagulopathy, hypertension, congenital heart disease)

For evaluation of neurologic symptoms or deficits[14]

~~(ACR, 2012a) Wippold~~

- Acute, new, or fluctuating neurologic symptoms or deficits such as, sensory deficits, limb weakness, **abnormal reflexes,** speech difficulties, visual loss*, lack of coordination, or mental status changes (
- ~~* Not explained by underlying ocular diagnosis, glaucoma, or macular degeneration~~**see background)**

For evaluation of known or suspected stroke or vascular disease[15-17]

~~(ACR 2012a, 2017a, 2019; Jauch, 2013)~~

- Known or suspected stroke with any acute, new, or fluctuating symptoms or deficits such as sensory deficits, limb weakness, speech difficulties, visual loss*, lack of coordination, or mental status changes (
- ~~*Not explained by underlying ocular diagnosis, glaucoma, or macular degeneration~~**see background)**

- Suspected stroke with a personal or first-degree family history (brother, sister, parent, or child) of aneurysm or known coagulopathy or on anticoagulation
- Symptoms of transient ischemic attack (TIA) (episodic neurologic symptoms such as sensory deficits, limb weakness, speech difficulties, visual loss, lack of coordination, or mental status changes)
- Evaluation of suspected acute subarachnoid hemorrhage (SAH)
- Follow-up for known hemorrhage, hematoma, or vascular abnormalities
  Note: MRI is the study of choice for detecting cavernous malformations (CCM) and other low flow vascular malformations (see background). Follow-up imaging of known CCM should be done only to guide treatment decisions or to investigate new symptoms. First-degree relatives of patients with more than one family member with a CCM should have a screening MRI as well as genetic counseling[18-20] ~~(Akers, 2017; Velz, 2018; Zyck, 2021)~~
- Suspected central venous thrombosis - see background[15, 21] ~~(ACR, 2017a, Bushnell, 2014)~~
- 1-time screening for silent cerebral infarcts in school age children and adults with sickle cell disease[22]
- Evaluation of neurological signs or symptoms in sickle cell disease[23, 24] ~~(Mackin, 2014; Thust, 2014)~~
- High stroke risk in sickle cell patients (2 - 16 years of age) with a transcranial doppler velocity > 200[25, 26]

For evaluation of known or suspected trauma[27-29]
~~(ACR, 2019f, Jagoda, 2008; Polinder, 2018)~~

- Known or suspected trauma or injury to the head with documentation of one or more of the following acute, new, or fluctuating:
  - Focal neurologic findings
  - Motor changes
  - Mental status changes
  - Amnesia
  - Vomiting
  - Seizures
  - Headache
  - Signs of increased intracranial pressure
- Known coagulopathy or on anticoagulation
- Known or suspected skull fracture by physical exam and/or prior imaging
- Post concussive syndrome if persistent or disabling symptoms and imaging has not been performed
- Subacute or chronic traumatic brain injury with new cognitive and/or neurologic deficit

For evaluation of suspected brain tumor, mass, or metastasis[30, 31]
~~(Kerjnick, 2008; NCCN, 2020)~~

- Suspected brain tumor with any acute, new, or fluctuating neurologic symptoms or deficits such as sensory deficits, limb weakness, abnormal reflexes, speech difficulties, visual loss*, lack of coordination, or mental status changes (

- ~~* Not explained by underlying ocular diagnosis, glaucoma, or macular degeneration~~see background)
-
- Suspected brain metastasis or intracranial involvement in patients with a history of cancer based on neurological symptoms or examination findings (may include new or changing lymph nodes)
-
- Histiocytic Neoplasms ~~(Erdheim-Chester Disease, Langerhans Cell Histiocytosis, and Rosai-Dorfman Disease)~~ for screening and/or with neurological signs or symptoms[32, 33] ~~27~~
  - Erdheim-Chester Disease
  - Langerhans Cell Histiocytosis
  - Rosai-Dorfman Disease)
- Midline dermoid cysts/sinuses with concern for intracranial extension[34-37]

~~Langerhans cell histiocytosis with visual, neurological, or endocrine abnormality; polyuria or polydipsia; suspected craniofacial bone lesions, aural discharge, or suspected hearing impairment/mastoid involvement[27] (Haupt, 2013; NCCN, 2020)~~

- Suspected Pituitary Tumors[38-41]
  - ~~(ACR, 2018; GHRS, 2000; Kannan, 2013; Majumdar, 2013)~~
  - ~~With the following:~~
  - Neurologic findings (e.g., visual field deficit suggesting compression of the optic chiasm, diplopia, gaze palsy)
  - Suspected ~~hypofunctioning~~hypofunctioning pituitary gland based on hormonal testing~~, e.g.,~~
    - ~~hypopituitarism~~Hypopituitarism~~,~~
    - ~~growth~~ Growth hormone deficiency~~,~~
    - ~~hypogonadotropic~~ Hypogonadotropic hypogonadism [~~i.e.,~~ low sex hormones and gonadotropins (FSH/LH) ~~and sex hormones*~~][42]
    -
      - ~~* Severe secondary hypogonadism with t~~Total testosterone persistently < 150 ~~and~~ with low or normal LH/FSH i.e., severe secondary hypogonadism **OR**
      - Total ~~* t~~Testosterone levels persistently ~~below normal range~~borderline around the lower limits of normal range (200-400 ng/dL) with low or normal LH/FSH; **AND**
        - Neurological signs ~~and~~ or symptoms; OR
        - Other pituitary hormonal abnormalities; OR
        - Low free testosterone and ~~C~~consideration of reversible functional causes of gonadotropin suppression (e.g., obesity, opioid use, diabetes, steroid use, or comorbid illness)
  - Suspected hyperfunctioning pituitary gland based on hormonal testing~~, i~~
    - ~~.e., c~~Central hyperthyroidism (high TSH)~~,~~
    - Cushing disease (high ACTH)
    - ~~, a~~Acromegaly/gigantism (high GH/IGF-1)
    - ~~or E~~elevated prolactin[43-45]
      - ~~(~~≥ 250 ng/mL ~~or~~ OR

- In the absence of another cause, e.g., stress, pregnancy, hypothyroidism, renal insufficiency, medication
  - > 100 ng/mL OR
  - ~~P~~persistently elevated OR
  -
  - ~~N~~neuroendocrine signs or symptoms (i.e., headache, galactorrhea, abnormal menses, infertility, or bitemporal hemianopsia)~~persistently elevated in the absence of another cause, e.g., stress, pregnancy, hypothyroidism, medication)~~
  -
- Central Diabetes Insipidus (low ADH)
- Precocious puberty in a child (male < 9; female < 8), with hormonal studies suggesting a central cause ~~and evidence of an accelerated bone age on x-ray~~ y[46] ~~(Faizah, 2012)~~
- Pituitary apoplexy with sudden onset of neurological and hormonal symptoms

- For screening for known non-CNS Cancer[47-56] - see **background** ~~(NCCN, 2020)2021 2022~~
  - Default screening for
    - Kidney cancer
    - Lung cancer
    - Merkel cell carcinoma
    - Mucosal melanoma of the head and neck, especially of the oral cavity
    - Poorly differential neuroendocrine cancer (Large or Small cell/Unknown primary of neuroendocrine origin)
  - Screening with preconditions
    - AML…… Suspicion of leukemic meningitis
    - Cutaneous melanoma…….. Stage IIIC or higher
    - Testicular cancer-Seminoma…….. High risk
    - Gestational Trophoblastic Neoplasia…….. Pulmonary metastasis
    - Bladder cancer…… High risk, i.e., small cell
  - All other cancer if CNS symptoms present
- For screening of Hereditary Cancer Syndromes - see **background**
  - Li Fraumeni syndrome- Annually[57]~~42~~
  - Von Hippel Lindau – Every 2 years, starting at age of 8 years[58] ~~43~~
  - Tuberous Sclerosis – Every 1-3 years, until the age of 25 years[59]~~44~~
  - MEN1 – Every 3-5 years, starting at the age of 5 years[60]~~45~~
  - NF-2- Brain IAC: Annually starting at the age of 10 years[61]~~46~~
  - Sturge Weber Syndrome: Once, after age 1 to rule out intracranial involvement; in patients <1 year, only if symptomatic[62]~~47~~

For evaluation of known brain tumor, mass, or metastasis

- Follow-up of known CNS cancer (either primary malignant brain tumor or secondary brain metastasis) ~~malignant brain tumor~~as per NCCN[31]
- Suspected recurrence with prior history of CNS cancer based on neurological symptoms or examination findings

- ~~Patient with history of CNS cancer (either primary or secondary) and a recent course of chemotherapy, radiation therapy (to the brain), or surgical treatment within the last two (2) years (NCCN, 2020)~~
- Follow-up of known ~~non-malignant brain~~**low grade** tumor **(WHO I-II) (i.e., meningioma, glioma, astrocytoma, oligodendroglioma)**~~/~~
  - **For surveillance as per NCCN**[31]
  - ~~lesion if~~**If** symptomatic, new/changing signs or symptoms or complicating factors
- ~~Follow-up of known meningioma[63] (NHS, 2018)~~
  - ~~If <2cm or heavily calcified at 2 years and 5 years~~
  - ~~> 2cm annually for 3 years and then scans at 5 years and 10 years~~
  - ~~Multiple meningiomas, annually~~
  - ~~After treatment (surgery or radiotherapy), post-operative if concern for residual tumor, every 6-12 months, then annually for 3-5 years based on WHO Grade (see background)~~
- Follow-up of known pituitary adenoma
  - New neuroendocrine signs or symptoms
  - Functioning adenoma - to assess response to treatment and 1-year follow-up after drug holiday[63] ~~(Stoller, 2015)~~
  - **Asymptomatic** ~~Asymptomatic~~ Macroadenoma (≥ 10mm) follow-up every 6-18 months, post-surgical follow-up **every** 1-2 years after surgery[64] ~~(Dekkers, 2008)~~
  - Asymptomatic, non-functioning Microadenoma < 10mm repeat in one year; if stable, repeat every 2-3 years[65] ~~(Lake, 2013)~~
- Follow-up of known pineal cyst (≥ 5mm) if there are atypical features or symptoms (e.g., headaches, gaze paresis, ataxia, papilledema, nausea/vomiting)[66, 67] ~~(Cauley, 2009; Jussila 2017)~~
- Follow-up of known arachnoid cyst[68-70] ~~(Al-Holou, 2010, 2013; Mustansir, 2018)~~
  - < 4 years old, serial imaging is warranted
  - > 4 years old, repeat imaging only if newly symptomatic, i.e., headaches, increased intracranial pressure, hydrocephalus, local mass effect, seizures, visual/endocrine dysfunction
- Tumor ~~evaluation and~~ monitoring in neurocutaneous syndromes ~~– see background~~**as per tumor type**
-
- **Histiocytic Neoplasms t**~~T~~o assess treatment response and surveillance of known brain lesions[32, 33, 71]
  - **Erdheim-Chester Disease**
  - **Langerhans Cell Histiocytosis**
  - **Rosai-Dorfman Disease**
- ~~(Erdheim-Chester Disease, Langerhans Cell Histiocytosis, and Rosai-Dorfman Disease)Langerhans cell histiocytosis[32, 33, 71] (Haupt, 2013, NCCN, 2020)~~
- ~~To assess treatment response and surveillance of known brain lesions~~

~~For screening for known Non-CNS Cancer - see background~~
~~(NCCN, 2020)~~

- ~~Default screening for~~
  - ~~Kidney cancer~~
  - ~~Lung cancer~~

- ~~Merkel cell carcinoma~~
- ~~Mucosal melanoma of the head and neck, especially of the oral cavity~~
- ~~Poorly differential neuroendocrine cancer (Large or Small cell/Unknown primary of neuroendocrine origin)~~

- ~~Screening with preconditions~~
  - ~~AML…… Suspicion of leukemic meningitis~~
  - ~~Cutaneous melanoma…… Stage IIIC or higher~~
  - ~~Testicular cancer-Seminoma…… High risk~~
  - ~~Gestational Trophoblastic Neoplasia…… Pulmonary metastasis~~
  - ~~Bladder cancer…… High risk, i.e., small cell~~
- ~~All other cancer if CNS symptoms present~~

~~**For screening of Hereditary Cancer Syndromes**~~

- ~~Li Fraumeni syndrome- Annually[42] (Kumar, 2018)~~
- ~~Von Hippel Lindau – Every 2 years, starting at age of 8 years[43] (Rednam, 2017)~~
- ~~Tuberous Sclerosis – Every 1-3 years, until the age of 25 years[44] (Krueger, 2013)~~
- ~~MEN1 – Every 3-5 years, starting at the age of 5 years[45] (Brandi, 2001)~~
- ~~NF-2- Brain IAC: Annually starting at the age of 10 years[46] (Evans, 2017)~~
- ~~Sturge Weber Syndrome: Once, after age 1 to rule out intracranial involvement; in patients <1 year, only if symptomatic[47] (Comi, 2011)~~

Indications for combination studies for the initial pre-therapy staging of cancer, OR active monitoring for recurrence as clinically indicated, OR evaluation of suspected metastases[31]
~~(NCCN, 2020)~~

- ≤ 5 concurrent studies to include CT or MRI of any of the following areas as appropriate depending on the cancer: Neck, Abdomen, Pelvis, Chest, Brain, Cervical Spine, Thoracic Spine or Lumbar Spine

For evaluation of known or suspected seizure disorder[72-77]
~~(ACR, 2019d; Cendes, 2016; Gaillard, 2009; Ho, 2013; Krumholz, 2007; Ramli, 2015)~~

- New onset of an unprovoked seizure in adults
- Newly identified change in seizure activity/pattern
- Known seizure disorder without previous imaging
- Medically refractory epilepsy
- Imaging indications for new onset seizures in the pediatric population[78-81] ~~(Hirtz, 2000; Kimia, 2012; Sadeq, 2016; Shaikh, 2019)~~
  - Abnormal neurological exam, especially a postictal focal deficit
  - Significant developmental delay
  - Focal onset
  - EEG shows focal or suspected structural abnormalities
  - <1 year of age

Note: Imaging is not indicated in simple febrile seizures

For evaluation of suspected multiple sclerosis (MS)[82-85]
~~(CMSC, 2018; Thompson, 2017; Traboulsee, 2016)~~

- For evaluation of patient with neurologic symptoms or deficits suspicious for MS with
  - A clinically isolated syndrome (optic neuritis, transverse myelitis, or brain stem syndrome); **OR**

- Recurrent episodes of variable neurological signs or symptoms not attributable to another cause

- To demonstrate dissemination in time for diagnosis (every 6-12 months~~) for high risk, 12-24 months for low risk)~~

For evaluation of known multiple sclerosis (MS)[82, 85, 86]
~~**(CMSC, 2018)**~~

- To establish a new baseline (no recent imaging, postpartum, or ~~6-12~~3-6 months after switching disease modifying therapy)
- Prior to starting or switching disease-modifying therapy
- **6-month repeat scan in patients with MRI disease activity that is not associated with clinical activity on a follow-up scan**
- ~~Prior to starting or switching disease-modifying therapy~~
- Every 1-2 years while on disease-modifying therapy to assess for subclinical disease activity, less frequently when stable for 2-3 years
- New signs or symptoms suggested of an exacerbation or unexpected clinical worsening
- Progressive Multifocal Leukoencephalopathy (PML) surveillance for patients on natalizumab (Tsyabri)[87] ~~(McGuigan, 2016)~~
    - 12 months after the start of treatment in all patients
    - Further surveillance MRI scanning timing is based on ~~anti-JCV antibody status~~**risk**
        - **Annually, i**~~I~~if anti-JCV antibody negative, ~~annually~~
        - ~~If anti-JCV antibody positive and antibody index < 1.5, every 6 months~~
        - ~~If anti-JCV antibody positive and antibody index > 1.5, e~~Every 3-4 months**, if high risk of PML occurrence:**
            - **seropositive for JC virus and have been treated with natalizumab for ≥18 months ~~or~~OR**
            - **high anti-JC virus antibody index values (>0.9) ~~or~~OR**
            - **previously treated with immunosuppressive therapies**
    - **Brain MRI every 3–4 months for up to 12 months, in high-risk patients who switch from natalizumab to other therapeutics**

Note: In the pediatric population, use a similar scan frequency for disease and therapeutic monitoring. Increase frequency of imaging (e.g., every 6 months) in children with highly active disease or in situations where imaging will change management.

For evaluation of known or suspected infectious or inflammatory disease (e.g., meningitis or abscess)[88, 89]
~~(Lummel, 2016; Oliveira, 2014)~~

- Suspected intracranial abscess or brain infection with acute altered mental status or with positive lab findings (such as elevated WBCs) OR follow-up assessment during or after treatment completed
- Meningitis with positive signs and symptoms (such as fever, headache, mental status changes, stiff neck) OR with positive lab findings (such as elevated white blood cells or abnormal lumbar puncture fluid exam)

- Suspected encephalitis with headache and altered mental status or follow-up as clinically warranted
- Endocarditis with suspected septic emboli
- Suspected temporal arteritis in a patient ≥ 50 with temporal headache, abrupt visual changes, jaw claudication, temporal artery tenderness, constitutional symptoms or elevated ESR ~~(Diamantopoulos, 2014; D'Souza, 2016; Klink, 2014; Salehi, 2016; Yip 2020)~~;[90-94] **AND**
    - Negative initial work-up (color Doppler ultrasonography or biopsy); **OR**
    - Atypical features, failure to response to treatment or concern for intracranial involvement

  Note: Protocol should include high-resolution contrast-enhanced imaging the temporal artery
- Central Nervous System (CNS) involvement in patients with known or suspected vasculitis or autoimmune disease with abnormal inflammatory markers or autoimmune antibodies
- Suspected primary CNS vasculitis based on neurological signs and symptoms with completed infectious/inflammatory lab work-up[95, 96] ~~(Godasi, 2019; Zuccoli, 2011)~~
- Immunocompromised patient (e.g., transplant recipients, HIV with CD4<200, primary immunodeficiency syndromes, hematologic malignancies) with focal neurologic symptoms, headaches, behavioral, cognitive or personality changes
- **Neurosarcoid**[97-99]
    - **Initial Evaluation:**
        - **Suspected based on neurological sign/symptoms and lab work (ACE, CSF analysis) OR**
        - **Known history of sarcoidosis with neurological signs or symptoms**
    - **Follow-up of known neurosarcoidosis:**
        - **To assess treatment response**
        - **Worsening signs or symptoms**
- ~~•~~

For evaluation of clinical assessment documenting cognitive impairment of unclear cause[100-102] ~~(Harvey 2012; HQO, 2014; Narayanan, 2016)~~

- Mental status score of either MMSE or MoCA of less than 26 or other similar mental status instruments*/formal neuropsychological testing showing at least mild cognitive impairment AND a completed basic metabolic workup (such as thyroid function testing, liver function testing, complete blood count, electrolytes, and B12)

*Other examples include: **Mini-Cog, Memory Impairment Screen, Saint Louis University Mental Status Examination (SLUMS), Brief Alzheimer's Screen (BAS), Blessed Dementia Scale (BDS), Clinical Dementia Rating (CDR)**~~Ottawa 3DY (O3DY), Brief Alzheimer's Screen (BAS), Blessed Dementia Scale (BDS), caregiver-completed AD8 (cAD8), Brief Cognitive Rating Scale (BCRS), Clinical Dementia Rating (CDR)~~[103, 104] ~~(Carpenter, 2011; McDougall, 1990)~~

FDA labeling for the drug Aduhelm (for Alzheimer's disease) requires baseline imaging and monitoring with Brain MRI.[105] Criteria for coverage includes the following:

- ~~o~~ Baseline study within 1 year of initiating treatment unless the patient has a more recent exacerbation, traumatic event [e.g., falls, etc.], or co-morbidity necessitating an evaluation within one-month preceding initiation
- ~~o~~ Prior to the 7th and 12th infusions

- ~~o~~ Monitoring if radiographic severe Amyloid Related Imaging Abnormalities (ARIA) is suspected or observed

NOTE: Enhanced clinical vigilance for ARIA is recommended during the first 8 doses of treatment with Aduhelm, particularly during titration. If a patient experiences symptoms which could be suggestive of ARIA, clinical evaluation should be performed, including MRI testing if indicated.

For evaluation of movement disorders[106-111]
~~**(ACR, 2019e; Albanese, 2011; Mascalchi, 2012; McFarland, 2014; Pyatigorskaya, 2014; Sharifi, 2014)**~~

- For evaluation of suspected Parkinson's with atypical feature or unresponsive to levodopa
- For evaluation of new non-Parkinson ~~symptoms~~**neurological symptoms** in known Parkinson's disease complicating the evaluation of the current condition
- For the evaluation of other movement disorder to exclude a structural lesion (i.e., suspected Huntington disease, chorea, atypical parkinsonian syndromes, hemiballismus, atypical dystonia)
  Note: MRI not indicated in essential tremor**, Tourette' syndrome,** or isolated focal dystonia (e.g., blepharospasm, cervical dystonia, laryngeal dystonia, oromandibular dystonia, writer's dystonia)[107, 111, 112] ~~(Alabanese, 2011; Comella, 2019; Sharfi, 2014)~~

For evaluation of cranial nerve and visual abnormalities

- Anosmia (loss of smell) or dysosmia documented by objective testing that is persistent and of unknown origin[113-115] ~~(Decker, 2013; Policeni, 2017; Rouby, 2011)~~
- Optic neuritis
- Abnormal eye findings on physical or neurologic examination (papilledema, pathologic nystagmus, optic atrophy, ocular nerve palsies, new onset anisocoria, visual field deficit, etc.)[116] ~~(Chang, 2019)~~
  Note: ~~Not explained by underlying ocular diagnosis, glaucoma, or macular degeneration~~**See background**
- Binocular diplopia with concern for intracranial pathology[117] **after comprehensive eye evaluation**[118] ~~(Iliescu, 2017)~~
- Childhood strabismus with development delay or abnormal fundoscopic exam to rule out intracranial abnormalities[119, 120] ~~(Kadom, 2008; Yoon, 2019)~~
- Horner's syndrome with symptoms localizing the lesion to the central nervous system[121] ~~(Lee, 2007)~~
- Trigeminal neuralgia or ~~other trigeminal autonomic cephalgias~~**neuropathy**, notably ~~in those~~ with **an** atypical presentation[5, 122, 123] ~~(Bendtsen, 2019; Cruccu, 2016; Wilbrink, 2009)~~
- **Occipital Neuralgia to exclude a structural lesion, notably in atypical cases**[124-126]
- Bell's Palsy- if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset[127] ~~(Quesnel, 2010)~~
- Hemifacial spasm[128] ~~(Hermier, 2019)~~
- Other objective cranial nerve palsy (CN IX-XII)[114, 129] ~~(ACR, 2017b; Mumtaz, 2014; Policeni, 2017)~~
- Bulbar symptoms, i.e., difficulty in chewing, weakness of the facial muscles, dysarthria, palatal weakness, dysphagia, and dysphonia and/or signs, i.e., atrophy and fasciculations of the tongue and absent gag reflex[130] ~~(Yedavelli, 2018)~~
- Pseudobulbar symptoms, i.e., dysphagia, dysarthria, facial weakness, sudden, stereotyped emotional outbursts that are not reflective of mood and/or signs, i.e., spastic tongue and exaggerated gag/jaw jerk[131] ~~(King, 2013)~~

For evaluation of known or suspected congenital abnormality (such as~~-~~ craniosynostosis, neural tube defects)[132, 133] ~~(Ashwal, 2009; Vinocur, 2010)~~

- Known or suspected congenital abnormality with any acute, new, or fluctuating neurologic, motor, or mental status changes
- Evaluation of macrocephaly in an infant/child <18 with previously abnormal US, abnormal neurodevelopmental ~~examination (Tan, 2018),[100] signs~~**examination, signs** of increased ICP or closed anterior fontanelle[134]
- Evaluation of microcephaly in an infant/child < 18
- Evaluation of craniosynostosis and other skull deformities. CT is preferred imaging to assess bony structures; MRI imaging is preferred to assess intracranial soft tissue
- Evaluation of the corticomedullary junction in Achondroplasia[135, 136] ~~(Dougherty,2018; Kubota, 2020))~~
- **Cerebral palsy if etiology has not been established in the neonatal period, there is change in the expected clinical or developmental profile or concern for progressive neurological disorder[137, 138]**
- **X-linked Adrenoleukodystrophy[139]**
    - **Baseline MRI between 12 and 18 months old**
    - **Second MRI 1 year after baseline**
    - **MRI every 6 months between 3 and 12 years old**
    - **Annual MRI after 12 years old**
- Prior treatment OR treatment planned for congenital abnormality
  Note: For evaluation of known or suspected hydrocephalus please see section on CSF abnormalities.

Cerebral Spinal Fluid (CSF) Abnormalities

- Evaluation of suspected hydrocephalus with any acute, new, or fluctuating neurologic, motor, or mental status changes
- Known hydrocephalus†
- For initial evaluation of a suspected Arnold Chiari malformation†
- Follow-up imaging of a known type II or type III Arnold Chiari malformation‡. For Arnold Chiari type I, follow-up imaging only if new or changing signs/symptoms[140] ~~(Whitson, 2015)~~
- Initial evaluation for a known syrinx or syringomyelia†
- Known or suspected normal pressure hydrocephalus (NPH)[141] ~~(Damasceno, 2015)~~
    - With symptoms of gait difficulty, cognitive disturbance, and urinary incontinence
- Follow-up shunt evaluation[142-145] ~~(Kamenova, 2018; Pople, 2002, Reddy, 2014; Wetzel, 2018)~~
    - Post operativity if indicated based on underlying disease or pre-operative radiographic findings and/or
    - 6-12 months after placement and/or
    - With neurologic symptoms that suggest shunt malfunction
- Evaluation of known or suspected cerebrospinal fluid (CSF) leakage[146] ~~(Severson, 2019)~~
- Cisternography for intermittent and complex CSF rhinorrhea/otorrhea. CSF fluid should always be confirmed with laboratory testing (Beta-2 transferrin assay)[147, 148] ~~(Mantur, 2011; Selcuk, 2010)~~

- Suspected spontaneous intra-cranial hypotension with distinct postural headache (other symptoms include nausea, vomiting, dizziness, tinnitus, diplopia neck pain or imbalance)[149, 150] ~~(Gordon 2009; NORD, 2017).~~
- CSF flow study for evaluation and management of CSF flow disorders[151, 152] ~~(Bradley, 2016; Mohammad, 2019)~~
  †Often congenital, but can present later in life; or less commonly acquired secondary to tumor, stroke, trauma, infection, etc.[153] ~~(NORD, 2014)~~

Pre-operative/procedural evaluation for brain/skull surgery

- Pre-operative evaluation for a planned surgery or procedure

Post-operative/procedural evaluation

- A follow-up study may be needed to help evaluate a patient's progress after treatment, procedure, intervention, or surgery. Documentation requires a medical reason that clearly indicates why additional imaging is needed for the type and area(s) requested.

Other Indications for a Brain MRI

- Vertigo associated with any of the following[154-156] ~~(Kattah, 2009; Welgampola, 2019; Yamada, 2019)~~
  - Signs or symptoms suggestive of a CNS lesion (ataxia, visual loss, double vision, weakness, or a change in sensation)
  - Progressive unilateral hearing loss
  - Risk factors for cerebrovascular disease with concern for stroke
  - After full neurologic examination and vestibular testing with concern for central vertigo (i.e., skew deviation, vertical nystagmus, head thrust test, videonystagmography (VNG)/ electronystagmography (ENG))
- Diagnosis of central sleep apnea on polysomnogram
  - Children > 1 year[157] ~~(Felix, 2016)~~
  - Adults in the absence of heart failure, chronic opioid use, high altitude, or treatment emergent central sleep apnea AND concern for a central neurological cause (Chiari malformation, tumor, infectious/inflammatory disease) OR with an abnormal neurological exam[158] ~~(Malhotra, 2010)~~
- Syncope with clinical concern for seizure or associated neurological signs or symptoms[159, 160] ~~(Al-Nsoor, 2010; Strickberger, 2006)~~
- Cyclical vomiting syndrome or abdominal migraine with any localizing neurological symptoms[161-163] ~~(Angus-Leppan, 2018; Li, 2018; Thangam, 2019)~~
- Soft tissue mass of the head with nondiagnostic initial evaluation (ultrasound and/or radiograph)[164-166] ~~(ACR, 2017c; Kim, 2019; Zhang, 2018)~~
- Psychological changes with neurological deficits on exam or after completion of a full neurological assessment that suggests a possible neurologic cause[167] ~~(ACR, 2019b)~~
- Global developmental delay or developmental delay with abnormal neurological examination in a child < 18 years[168, 169] ~~(Ali, 2015; Momen, 2011)~~
- ~~Cerebral palsy if etiology has not been established in the neonatal period, there is change in the expected clinical or developmental profile or concern for progressive neurological disorder[133, 134] (Ashwal, 2004; NICE, 2020)~~

- Unexplained event (BRUE) formerly apparent life-threatening event (ALTE) in infants < 1 year with concern for neurological cause based on history and exam[170] ~~(Tieder, 2016)~~
  Note: Imaging is not indicated in low-risk patients

Indications for a Brain MRI with Internal Auditory Canal (IAC)

- Unilateral non-pulsatile tinnitus
- Pulsatile tinnitus
- Suspected acoustic neuroma (Schwannoma) or cerebellar pontine angle tumor with any of the following signs and symptoms: unilateral hearing loss by audiometry, headache, disturbed balance or gait, unilateral tinnitus, facial weakness, or altered sense of taste
- Suspected cholesteatoma
- Suspected glomus tumor
- Asymmetric sensorineural hearing loss on audiogram
- Congenital/childhood sensorineural hearing loss suspected to be due to a structural abnormality[171-173] (CNVIII, the brain parenchyma, or the membranous labyrinth). CT is the preferred imaging modality for the osseous anatomy and malformations of the inner ear.
- CSF otorrhea (MRI for intermittent leak, CT for active leaks)[174]; ~~(Hiremath, 2019)~~ CSF fluid should always be confirmed with laboratory testing (Beta-2 transferrin assay)
- Clinical suspicion of acute mastoiditis as a complication of acute otitis media with intracranial complications (i.e., meningeal signs, cranial nerve deficits, focal neurological findings, altered mental status)[175, 176] ~~(Patel, 2014; Platzek, 2014)~~
- ~~Bell's Palsy for evaluation of the extracranial nerve course -if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset[127] (Quesnel, 2010)~~

Indications for Combination Studies[15, 16]

Note: These body regions might be evaluated separately or in combination as documented in the clinical notes by physical examination findings (e.g., localization to a particular segment of the neuroaxis), patient history, and other available information, including prior imaging.

Exception ~~:~~ ~~(ACR, 2017a, 2019a)~~

- ~~For~~ approved indications as noted above and being performed in a child under 8 years of age who will need anesthesia for the procedure and there is a suspicion of concurrent intracranial pathology[177] ~~(Lawson, 2000)~~

- **Brain MRI/Neck MRA***
  - Recent ischemic stroke or transient ischemic attack
  - Suspected carotid or vertebral artery dissection with focal or lateralizing neurological deficits

- **Brain MRI/Brain MRA***
  - Recent ischemic stroke or transient ischemic attack

- Thunderclap headache with continued concern for underlying vascular abnormality after initial negative **brain imaging > 6 hours after onset** ~~work-up~~[178-180] ~~(Whitehead, 2019, Yeh, 2010, Yuan, 2005)~~ ~~:~~ ~~brain imaging > 6 hours after onset~~
  Note: Negative brain CT < 6 hours after headache onset excludes subarachnoid hemorrhage in neurologically intact patients[181]
- ~~Negative Brain CT; **AND**~~
- ~~Negative Lumbar Puncture~~
- Acute, sudden onset of headache with personal history of a vascular abnormality or first-degree family history of aneurysm
- Headache associated with exercise or sexual activity[6] ~~(IHS, 2018)~~
- Suspected venous thrombosis (dural sinus thrombosis) – Brain MRV see **background**
- **Neurological signs or symptoms in sickle cell patients**
- **High stroke risk in sickle cell patients (2 - 16 years of age) with a transcranial doppler velocity > 200** [24]

- **Brain MRI/Brain MRA/Neck MRA***
  - Recent stroke or transient ischemic attack (TIA)
  - Suspected carotid or vertebral artery dissection with focal or lateralizing neurological deficits

- **Brain MRI with IAC/ Brain MRA/Neck MRA (any combination)***
  - **Pulsatile tinnitus with concern for a suspected arterial vascular and/or intracranial etiology**[182, 183~~12~~]

***Note: MRA and CTA are generally comparable noninvasive imaging alternatives each with their own advantages and disadvantages. Brain MRI can alternatively be combined with Brain CTA/Neck CTA.**

- ~~**Brain MRI/ Cervical MRI/Thoracic MRI (any combination)**~~
  - ~~For evaluation of neuromyelitis optica spectrum disorders (recurrent or bilateral optic neuritis; recurrent transverse myelitis)[143] (Wingerchuk, 2015)~~
  - ~~For known MS, prior to the initiation or change of disease modification treatments and assess disease burden (to establish a new baseline)~~
  - ~~Follow-up scans for known MS if patients have known spine disease[144] (Kaunzner, 2017)~~
    - ~~6-12 months after starting/changing treatment~~
      ~~Every 1-2 years while on disease-modifying therapy to assess for subclinical disease activity, less frequently when stable for 2-3 years~~
- **Brain MRI/-Cervical MRI/Thoracic MRI (any combination)**
  - **Combination studies for MS: These body regions might be evaluated separately or in combination as guided by physical examination findings (e.g., localization to a particular segment of the spinal cord), patient history (e.g., symptom(s), time course, and where in**

the CNS the likely localization(s) is/are), and other available information, including prior imaging.
    - ~~o~~ For evaluation of neuromyelitis optica spectrum disorders (recurrent or bilateral optic neuritis; recurrent transverse myelitis)[184]~~143~~
    - ~~o~~ For known MS, prior to the initiation or change of disease modification treatments and assess disease burden (to establish a new baseline)[185]~~144~~
    - ~~o~~ Follow-up scans, including brain and spine imaging, if patients have known spine disease:
        - ~~▪~~ 6-12 months after starting/changing treatment
        - ~~▪~~ Every 1-2 years while on disease-modifying therapy to assess for subclinical disease activity, less frequently when stable for 2-3 years
    - ~~▪~~

- **Brain MRI/~~-~~Cervical MRI/Thoracic MRI/Lumbar MRI (any combination)**
    - For initial evaluation of a suspected Arnold Chiari malformation
    - Follow-up imaging of a known type II or type III Arnold Chiari malformation~~‡~~. For Arnold Chiari type I, follow-up imaging only if new or changing signs/symptoms[140, 186] ~~(Radic, 2018; Whitson, 2015)~~
    - Oncological Applications (e.g., primary nervous system, metastatic)
        - Drop metastasis from brain or spine~~–~~ (see background)
        - Suspected leptomeningeal carcinomatosis (see background)[187]
        - Tumor evaluation and monitoring in neurocutaneous syndromes - See background
    - ~~Suspected Leptomeningeal carcinomatosis (see background)[146] (Shah, 2011)~~
    - ~~Tumor evaluation and monitoring in neurocutaneous syndromes - See background~~
    - CSF leak highly suspected and supported by patient history and/or physical exam findings (known or suspected spontaneous (idiopathic) intracranial hypotension (SIH), post lumbar puncture headache, post spinal surgery headache, orthostatic headache, rhinorrhea or otorrhea, or cerebrospinal-venous fistula)

- **Brain MRI/Orbit MRI**
    - Optic neuropathy or unilateral optic disk swelling of unclear etiology to distinguish between a compressive lesion of the optic nerve, optic neuritis, ischemic optic neuropathy (arteritic or non-arteritic), central retinal vein occlusion or optic nerve infitrative disorders[188] ~~(Behbehani, 2007)~~
    - Bilateral optic disk swelling (papilledema) with visual loss[189] ~~(Margolin, 2019)~~
    - Optic Neuritis
        - If atypical presentation (bilateral, absence of pain, optic nerve hemorrhages, severe visual impairment, lack of response to steroids, poor recovery or recurrence)[190, 191] ~~, severe visual impairment, or poor recovery following initial onset or treatment onset~~ ~~83~~ ~~(CMSC, 2018)~~
        - If needed to confirm optic neuritis and rule out compressive lesions
    - Known or suspected neuromyelitis optica spectrum disorder with severe, recurrent, or bilateral optic neuritis[184] ~~(Wingerchuk, 2015)~~

- **Brain MRI/FACE/SINUS/NECK MRI**
  - Anosmia or dysosmia on objective testing that is persistent and of unknown origin[113, 114, 192] ~~(Decker, 2013; Policeni, 2017; Zaghouani, 2013)~~
  - ~~Granulomatosis with polyangiitis (Wegener's granulomatosis) disease[193] (Pakalniskis, 2015)~~
  -
  - Trigeminal ~~Neuralgia~~ **neuralgia or** ~~or other trigeminal autonomic cephalgias~~**neuropathy**~~, notably in those~~ with **an** atypical presentation **(for evaluation of the extracranial nerve course)** [114, 194] ~~(Hughes, 2016; Policeni, 2017)~~
  - **Bell's Palsy/hemifacial spasm for evaluation of the extracranial nerve course -if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset**[127] ~~93~~
  - ~~Bells/hemifacial spasm that meets above criteria~~
  - Objective cranial nerve palsy (CN IX-XII) (for evaluation of the extracranial nerve course)[114, 129] ~~(Mumtaz, 2014; Policeni, 2017)~~

BACKGROUND

Brain (head) MRI is the procedure of choice for most brain disorders. It provides clear images of the brainstem and posterior brain, which are difficult to view on a CT scan. It is also useful for the diagnosis of demyelinating disorders (such as multiple sclerosis (MS) that cause destruction of the myelin sheath of the nerve). The evaluation of blood flow and the flow of cerebrospinal fluid (CSF) is possible with this non-invasive procedure.

MRI for Headache – Generally, magnetic resonance imaging is the preferred imaging technique for evaluating the brain parenchyma, and CT is preferable for evaluating subarachnoid hemorrhage. CT is faster and more readily available than MRI and is often used in urgent clinical situations. Neurologic imaging is warranted in patients with headache disorders along with abnormal neurologic examination results or predisposing factors for brain pathology. Contrast-enhanced MRI is performed for evaluation of inflammatory, infectious, neoplastic, and demyelinating conditions.

Headache timeframes and other characteristics – Generally, acute headaches are present from hours to days, subacute from days to weeks and chronic headaches for more than 3 months. Acute severe headaches are more likely to be pathological (e.g., SAH, cerebral venous thrombosis) than non-acute (e.g., migraine, tension-type). Headaches can also be categorized as new onset or chronic/recurrent. Non-acute new onset headaches do not require imaging unless there is a red flag as delineated above. Incidental findings lead to additional medical procedures and expense that do not improve patient well-being. Primary headache syndromes, such as migraine and tension headaches, are often episodic with persistent or progressive headache not responding to treatment requiring further investigation (e.g., new daily persistent headache). Imaging is indicated in chronic headaches if there is a change in the headache frequency (number of headaches episodes/month), duration of each episode, severity of the headaches or new characteristics, such as changing aura or associated symptoms.[1, 6, 195-197] ~~(ACR, 2019c; IHS, 2018; Jang, 2019; Spierings, 2003; Tyagi, 2012)~~

Migraine with aura[6, 7, 198] ~~(Hadjikhani, 2019; IHS, 2018; Micieli, 2020)~~ – The headache phase of a migraine is preceded and/or accompanied by transient neurological symptoms referred to as aura in at least a third of migraine attacks. The most common aura consists of positive and/or negative visual phenomena, present in up to 99% of the ~~patient~~**individual**s. Somatosensory is the secondary most common type of aura (mostly paraesthesias in an upper limb and/or hemiface). Language/speech (mainly paraphasia and anomic aphasia) can also be affected. These neurological symptoms typically evolve over a period of minutes and may last up to 20 minutes or more. The gradual evolution of symptoms is thought to reflect spreading of a neurological event across the visual and somatosensory cortices. Characteristically, the aura usually precedes and terminates prior to headache, usually within 60 minutes. In others, it may persist or begin during the headache phase. ICHD-3 definition of the aura of migraine with typical aura consists of visual and/or sensory and/or speech/language symptoms, but no motor, brainstem or retinal symptoms and is characterized by gradual development, duration of each symptom no longer than one hour, a mix of positive and negative features and complete reversibility. Atypical or complex aura includes motor, brainstem, monocular visual disturbances, or ocular cranial nerve involvement (hemiplegic migraine, basilar migraine/brainstem aura, retinal migraine, ophthalmoplegic migraine) and secondary causes need to be excluded. Additional features of an aura that raise concern for an underlying vascular etiology include late age of onset, short duration, evolution of the focal symptoms, negative rather than positive visual phenomenon, and history of vascular risk factors.

Neurological Deficits~~:~~ –
Examples of abnormal reflexes related to upper motor neuron lesion/central pathology~~:~~ include hyperreflexia, clonus, Hoffman sign and Babinski, snout, palmar grasp, and rooting reflexes.

Visual loss has many possible etiologies, and MRI is only indicated in suspected neurological causes of visual loss based on history and exam. Visual field defects, such as bitemporal hemianopsia, ~~or~~ homonymous hemianopsia, or quadranopsia, require imaging as well as does suspected optic nerve pathology. Subjective symptoms such as blurred vision or double vision with no clear correlate on neurological examination requires a comprehensive eye evaluation to exclude more common causes, such as cataracts, refractive errors, retinopathy, glaucoma, or macular degeneration. Transient visual loss with history consistent with TIA but normal exam at time of examination also should be imaged. Positive visual phenomena, such as photopsias or scintillations that march across the visual field, suggest migraine whereas negative phenomenon, such as shaded or blurred, is more characteristic of ischemia.

Table 1: Gait and brain imaging[199-204~~‡~~]

Gait	Characteristic	Work up/Imaging
Hemiparetic	**Spastic unilateral, circumduction**	**Brain and/or, Cervical spine imaging based on associated symptoms**
Diplegic	**Spastic bilateral, circumduction**	**Brain, Cervical and Thoracic Spine imaging**
Myelopathic	**Wide based, stiff, unsteady**	**Cervical and/or Thoracic spine MRI based on associated symptoms**

Ataxic	**Broad based, clumsy, staggering, lack of coordination, usually also with limb ataxia**	**Brain imaging**
Apraxic	**Magnetic, shuffling, difficulty initiating**	**Brain imaging**
Parkinsonian	**Stooped, small steps, rigid, turning en bloc, decreased arm swing**	**Brain Imaging**
Choreiform	**Irregular, jerky, involuntary movements**	**Medication review, consider brain imaging as per movement disorder Brain MR guidelines**
Sensory ataxic	**Cautious, stomping, worsening without visual input (ie + Romberg)**	**EMG, blood work, consider spinal (cervical or thoracic cord imaging) imaging based on EMG**
Neurogenic	**Steppage, dragging of toes**	**EMG, if there is foot drop, Lumbar spine MRI Pelvis MR appropriate evidence of plexopathy**
Vestibular	**Insecure, veer to one side, worse when eyes closed, vertigo**	**Consider Brain/IAC MRI as per GL**

~~([‡]References: Chhetri, 2014; Clinch, 2021; Gait, 2021; Haynes, 2018; Marshall, 2012; Pirker, 2017)~~

Non-neurological causes of gait dysfunction include pain (antalgic), side effects of drugs (analgesic, antihistamines, benzos, psych meds, antihypertensives), visual loss, hearing impairment, orthopedic disorders, rheumatologic disorders, psychogenic, and cardiorespiratory problems (orthostasis).[200, 202-204] ~~(Foster 2021; Haynes, 2018; Marshall, 2012; Pirker 2017).~~

MRI and recent stroke or transient ischemic attack – A stroke or central nervous system infarction is defined as "brain, spinal cord, or retinal cell death attributable to ischemia, based on neuropathological, neuroimaging, and/or clinical evidence of permanent injury. … Ischemic stroke specifically refers to central nervous system infarction accompanied by overt symptoms, whereas silent infarction causes no known symptoms."[205] If imaging or pathology is not available, a clinical stroke is diagnosed by symptoms persisting for more than 24 hours. Ischemic stroke can be further classified by the type and location of ischemia and the presumed etiology of the brain injury. These include large-artery atherosclerotic occlusion (extracranial or intracranial), cardiac embolism, small-vessel disease and less commonly dissection, hypercoagulable states, sickle cell disease and undetermined causes.[206] TIAs in contrast, "are a brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than one hour, and without evidence of acute infarction on imaging."[207] On average, the annual risk of future ischemic stroke after a TIA or initial ischemic stroke is 3–4%, with an incidence as high as 11% over the next 7 days and 24–29% over the following 5 years. This has significantly decreased in the last half century due to advances in secondary prevention.[208]

Therefore, when revascularization therapy is not indicated or available in individuals with an ischemic stroke or TIA, the focus of the work-up is on secondary prevention. This includes noninvasive vascular imaging to identify the underlying etiology, assess immediate complications and risk of future stroke. The majority of stroke evaluations take place in the inpatient setting. Admitting TIA patients is reasonable if they present within 72 hours and have an ABCD(2) score ≥3, indicating high risk of early recurrence, or the evaluation cannot be rapidly completed on an outpatient basis.[207] Minimally, both stroke and TIA should have an evaluation for high-risk modifiable factors, such as carotid stenosis atrial fibrillation as the cause of ischemic symptoms.[206] Diagnostic recommendations include neuroimaging evaluation as soon as possible, preferably with magnetic resonance imaging, including DWI; noninvasive imaging of the extracranial vessels should be performed, and noninvasive imaging of intracranial vessels is reasonable.[209]

Individuals with a history of stroke and recent work-up with new signs or symptoms indicating progression or complications of the initial CVA should have repeat brain imaging as an initial study. Individuals with remote or silent strokes discovered on imaging should be evaluated for high-risk modifiable risk factors based on the location and type of the presumed etiology of the brain injury.

~~**MRI and recent stroke or transient ischemic attack –** A stroke or central nervous system infarction is defined as "brain, spinal cord, or retinal cell death attributable to ischemia, based on neuropathological, neuroimaging, and/or clinical evidence of permanent injury. … Ischemic stroke specifically refers to central nervous system infarction accompanied by overt symptoms, whereas silent infarction causes no known symptoms" (Sacco, 2013).[206] If imaging or pathology is not available, a clinical stroke is diagnosed by symptoms persisting for more than 24 hours. Ischemic stroke can be further classified by the type and location of ischemia and the presumed etiology of the brain injury. These include large-artery atherosclerotic occlusion (extracranial or intracranial), cardiac embolism, small-vessel disease and less commonly dissection, hypercoagulable states, sickle cell disease and undetermined causes (Kernan, 2014).[207] TIAs in contrast, "are a brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than one hour, and without evidence of acute infarction on imaging" (Easton, 2009).[208] On average, the annual risk of future ischemic stroke after a TIA or initial ischemic stroke is 3–4%, with an incidence as high as 11% over the next 7 days and 24–29% over the following 5 years. This has significantly decreased in the last half century due to advances in secondary prevention (Hong, 2011).[209]~~

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~~Patients with a history of stroke and recent work-up with new signs or symptoms indicating progression or complications of the initial CVA should have repeat brain imaging as an initial study. Patients with remote or silent strokes discovered on imaging should be evaluated for high-risk modifiable risk factors based on the location and type of the presumed etiology of the brain injury.~~

Non-aneurysmal vascular malformations – Non-aneurysmal vascular malformations can be divided in low flow vascular malformations and high flow vascular malformations. Low flow vascular malformations include dural venous anomalies (DVA), cavernomas, and capillary telangiectasias. High flow vascular malformations include AVM and dural arteriovenous fistulas (dAVF). For low flow malformations, MRI is the study of choice. ~~There is L~~limited medical literature is available to support vascular imagining (CTA or MRA). CTA plays a limited role in the assessment of cavernoma but may be used to demonstrate a DVA. MRA is not usually helpful in the assessment of cavernoma, capillary telangiectasia, and DVA. Vascular imaging is indicated in high flow vascular malformations.[210-212]

MRI and Central Venous Thrombosis – a MR Venogram is indicated for the definite evaluation of a central venous thrombosis/dural sinus thrombosis. The most frequent presentations are isolated headache, intracranial hypertension syndrome **(headache, nausea/vomiting, transient visual obscurations, pulsatile tinnitus, CN VI palsy, papilledema),**[213] seizures, focal neurological deficits, and encephalopathy. Risk factors are hypercoagulable states inducing genetic prothrombotic conditions, antiphospholipid syndrome and other acquired prothrombotic diseases (such as cancer), oral contraceptives, pregnancy, puerperium (6-weeks postpartum), infections, and trauma. COVID-19 infection is associated with hypercoagulability, a thromboinflammatory response, and an increased incidence of venous thromboembolic events (VTE) ~~(Connors, 2020; Tu, 2020)~~.[214, 215] Since venous thrombosis can cause SAH, infarctions, and hemorrhage, parenchymal imaging with MRI/CT is also appropriate ~~(Bushnell, 2014; Courinho, 2015; Ferro, 2016)~~.[21, 216, 217]

~~**MRI and benign tumors** (e.g., schwannomas, choroid plexus papilloma, pineocytoma, gangliocytoma) A single follow-up study is appropriate after the initial diagnosis to ensure stability. Follow-up of known benign tumor is indicated if symptomatic, new/changing signs or symptoms, or complicating factors (Gupta, 2017).[165] In neurocutaneous and hereditary cancer syndromes, follow-up surveillance may also be indicated (see below).~~

Galactorrhea and MRI ~~–~~ ~~Imaging is not indicated in i~~Isolated galactorrhea without elevated prolactin (normoprolactinemic) ~~(Atluri, 2018; Huang, 2012)~~ **is.**[172, 173] **usually due to breast pathology, i.e., breast feeding, trauma, ill-fitting undergarments. Consider mammogram, breast ultrasound, and serial dilution of the ~~patient~~individual's prolactin sample to correct for possible hook effect.**[218, 219] ~~172, 173~~

~~**MRI and Meningioma**[63] (NHS, 2018) – For incidental meningiomas, most patients who progressed did so within 5 years of diagnosis (Islim, 2019).[221] Small (<2cm) meningiomas rarely grow sufficiently to produce symptoms within 5 years. Heavily calcified meningiomas rarely grow. Patients with multiple meningiomas should have annual scans indefinitely, despite treatment, because of the possibility of further meningiomas developing.~~

~~For surveillance post-treatment:~~

- ~~Solitary convexity WHO Grade 1 meningiomas - MRI scan at 2½ years post-operatively.~~
- ~~Solitary skull base or falcine origin WHO Grade 1 meningiomas- MRI scans at 1 year, 2 years, 3½ years and 5 years post-operatively. If a recurrence is detected, continue annual scans.~~
- ~~WHO Grade 2 meningiomas- MRI scan at 6 months, 1 year then annually to 5 years. If a recurrence is detected, continue annual scans.~~
- ~~WHO Grade 3 meningiomas - 6-monthly MRI scans for 3 years, then annual scans to 5 years. If a recurrence is detected, continue annual scans.~~
- ~~Patients who have had radiosurgery, including those being treated for a recurrence, should have scans at 6 months, then annually for 3 years, a scan at 5 years and a final scan at 10 years.~~

Table 2: MRI and staging screening in Non-CNS Cancers[48, 49, 51, 53] ~~(NCCN, 2020)~~

(NON-BRAIN/CNS) CANCER	PRECONDITION
Cutaneous melanoma	Stage IIIC or higher, default staging screening ≥ stage IIIC, surveillance with periodic brain MRI up to 3 years even if asymptomatic without prior brain mets; and if prior brain mets, surveillance every 3-6 months up to 3 years
Testicular cancer-Seminoma	If high risk, such as beta HCG >5000IU/L, or multiple lung or visceral mets, choriocarcinoma, neurological symptoms, or AFP>10,000ng/ml
Merkel cell carcinoma	Default staging screening, but especially for high risk (≥stage IIIb, immunosuppression)
Lung cancer	Default staging screening brain MRI also for surveillance in small cell every 3 months for 2 years if they have had no prophylactic cranial radiation

MRI and Neurocutaneous Syndromes

- In NF-1, clinical evaluation appears to be more useful to detect complications than is screening imaging in asymptomatic ~~patient~~**individual**s. Imaging is indicated in evaluation of suspected tumors based on clinical evaluation and for follow-up of known intracranial tumors ~~(Borofsky, 2013)~~.[220]
- Conversely in NF-2, routine MR imaging screening is always indicated, given the high prevalence of CNS tumors, especially vestibular schwannomas. In ~~patient~~**individual**s with NF-2, routine screening brain/IAC imaging is indicated annually starting from age 10 if asymptomatic or earlier with clinical signs/symptoms. Most individuals with NF2 eventually develop a spinal tumor, most commonly schwannomas, but meningioma and ependymomas are also seen. Spinal imaging at baseline and

every 2 to 3 years is also advised with more frequent imaging, if warranted, based on sites of tumor involvement ~~(Evans, 2017)~~.[61]

- In ~~patient~~**individual**s with Tuberous Sclerosis, Brain MRI should be obtained every 1-3 years up until age 25 for surveillance for CNS abnormalities ~~(Krueger, 2013)~~.[59]
- In Von Hippel Lindau Syndrome, imaging of the brain and spinal cord for hemangioblastomas is recommended every 2 years ~~(Rednam, 2017)~~.[58]
- In Sturge Weber Syndrome, Brain MRI can rule out intracranial involvement only after age 1 and is recommended in ~~patient~~**individual**s <1 year only if symptomatic ~~(Comi, 2011)~~.[62]

MRI and Positron Emission Tomography (PET) for Chronic Seizures – When MRI is performed in the evaluation of ~~patient~~**individual**s for epilepsy surgery, almost a third of those with electrographic evidence of temporal lobe epilepsy have normal MRI scans. Interictal positron emission tomography (PET) may be used to differentiate ~~patient~~**individual**s with MRI-negative temporal lobe epilepsy.

Multiple Sclerosis[83, 221, 222] ~~(Rovira, 2015; Saguil, 2014; Thompson, 2018)~~ – The diagnosis of MS requires demonstration of lesions in the CNS disseminated in time and space and the absence of fever, infection, or other more likely etiologies. ~~There is an~~**An** expanding amount of available disease-modifying treatments ~~that~~ are effective in slowing down disease progression, especially in the early stages. These treatments can have serious side effects and can be costly; therefore, the accurate and expeditious diagnosis of MS is critical.

The diagnosis of MS can be made on clinical presentation alone with 2 clinical attacks and objective clinical evidence of more than 2 lesions. Attacks may be ~~patient~~**individual**-reported or objectively observed and must last for a minimum of 24 hours and be 30 days apart. However, corroborating magnetic resonance imaging (MRI) is the diagnostic standard and is used, as well, to rule out other disorders. Additionally, MRI findings can replace certain clinical criteria in a substantial number of ~~patient~~**individual**s. In the revised McDonald Criteria, MRI findings can be used to establish dissemination in both time and space.

Table 3: Variable Symptoms and Signs of MS

Symptoms	***Signs***
Depressed mood	Ataxia
Memory loss/cognitive changes	Dysmetria
Dizziness or vertigo	Decreased sensation (pain, vibration, position)
Fatigue	Decreased strength
Hearing loss and tinnitus	Hyperreflexia, spasticity
Heat sensitivity (Uhthoff Phenomenon)	Nystagmus

Incoordination and gait disturbances	Lhermitte's sign
Sensory disturbances (dysesthesias, numbness, paresthesias)	Visual defects (internuclear ophthalmoplegia, optic disc pallor, red color desaturation, reduced visual acuity)
Pain	
Urinary symptoms	
Visual disturbances (diplopia, oscillopsia)	
Weakness	

In the presence of a clear, clinically isolated syndrome such as optic neuritis, transverse myelitis, or brain stem syndrome, brain MRI is the next diagnostic step. MS can also have variable and often subjective symptoms that come and go (see **Table 3**). If there are recurrent episodes of variable neurological signs or symptoms not attributable to another cause with clinical concern for MS, imaging is warranted as well.

MRI and Neuromyelitis optica spectrum disorders (NMOSD)[184] ~~(Wingerchuk, 2015)~~ ~~–~~ NMOSD are inflammatory disorders of the central nervous system characterized by severe, immune-mediated demyelination and axonal damage predominantly affecting the optic nerves and spinal cord, but also the brain and brainstem. NMOSD can be distinguished from multiple sclerosis and other inflammatory disorders by the presence of the aquaporin-4 (AQP4) antibody. Features of NMOSD include attacks of bilateral or sequential optic neuritis acute transverse myelitis and the area postrema syndrome (with intractable hiccups or nausea and vomiting). The evaluation of suspected NMOSD entails brain and spinal cord neuroimaging. In contrast to MS (in which spinal cord involvement tends to be incomplete and asymmetric), NMOSD have a longer extent of spinal cord demyelination generally involving three or more vertebral segments.

Temporal Arteritis ~~–~~ Giant cell arteritis (GCA) is an inflammatory disorder that should be considered in ~~patient~~**individual**s over the age of 50 with the following signs or symptoms: new headaches, acute onset of visual disturbances (especially transient monocular visual loss), jaw claudication, constitutional symptoms, tenderness over the temporal artery, and elevated ESR and/or CRP. A diagnosis of polymyalgia rheumatica (PMR) is highly associated. Large vessel GCA denotes involvement of the aorta and its first-order branches, especially the subclavian arteries, and is common. Extra- and intracranial cerebral vasculitis can also be seen, but is more rare, and strokes are related to vasculitis of extracranial cerebral arteries causing vertebral or internal carotid arteries stenosis. Gold standard for diagnosis of GCA is temporal artery biopsy. Color Doppler ultrasound (CDUS) can be used as a surrogate for temporal artery biopsy in some cases. High-resolution magnetic resonance imaging (MRI) can visualize the temporal arteries when used with contrast. The presence of clinical manifestations unusual in GCA should prompt consideration of alternative diagnoses. Examples of such include adenopathy, pulmonary infiltrates, digital cyanosis, ulceration or gangrene, mononeuritis multiplex,

stroke in the distribution of the middle cerebral artery, glomerulitis, and/or rapidly rising creatinine ~~(Diamantopoulos, 2014; D'Souza, 2016; Klink, 2014; Larivière, 2014; Salehi, 2016; Yip 2020)~~.[90-94, 223]

MMSE ~~–~~ The Mini Mental State Examination (MMSE) is a tool that can be used to systematically and thoroughly assess mental status. It is an 11-question measure that tests five areas of cognitive function: orientation, registration, attention and calculation, recall, and language. The MMSE has been the most commonly used measure of cognitive function in dementia research, but researchers have recognized that it is relatively insensitive and variable in mildly impaired individuals. The maximum score is 30. A score of 23 or lower is indicative of cognitive impairment. The MMSE takes only 5-10 minutes to administer and is, therefore, practical to use repeatedly and routinely.

MoCA ~~–~~ The Montreal Cognitive Assessment (MoCA) was designed as a rapid screening instrument for mild cognitive dysfunction. It assesses different cognitive domains: attention and concentration, executive functions, memory, language, visuoconstructional skills, conceptual thinking, calculations, and orientation. MoCA differs from the MMSE mainly by including tests of executive function and abstraction, and by putting less weight on orientation to time and place. Ten of the MMSE's 30 points are scored solely on the time-place orientation test, whereas the MoCA assigns it a maximum of six points. The MoCA also puts more weight on recall and attention-calculation performance, while de-emphasizing language skill. Time to administer the MoCA is approximately 10 minutes. The total possible score is 30 points; a score of 26 or above is considered normal.

MRI and Movement disorders ~~–~~ Atypical parkinsonian syndromes include progressive supranuclear palsy (PSP), multiple system atrophy (MSA), corticobasal degeneration (CBD), and dementia with Lewy bodies.

Anosmia ~~–~~ Nonstructural causes of anosmia include post-viral symptoms, medications (Amitriptyline, Enalapril, Nifedipine, Propranolol, Penicillamine, Sumatriptan, Cisplatin, Trifluoperazine, Propylthiouracil). These should be considered prior to advanced imaging to look for a structural cause.

Anosmia and dysgeusia have been reported as common early symptoms in ~~patient~~**individual**s with COVID-19, occurring in greater than 80 percent of ~~patient~~**individual**s. For isolated anosmia, imaging is typically not needed once the diagnosis of COVID has been made given the high association. As such, COVID testing should be done prior to imaging ~~(Geyer, 2008; Lechien, 2020; Saniasiaya, 2021)~~.[224-226]

Evaluation of olfactory function is essential to determine the degree of chemosensory loss and confirm the ~~patient~~**individual**'s complaint. It also allows monitoring of olfactory function over time, helps to detect malingerers, and to establish compensation for disability. ~~There are~~**The** two general types of olfactory testing~~:~~ **include** psychophysical and electrophysiologic testing. Psychophysical tests are used for clinical evaluation of olfactory loss; whereas, electrophysiologic tests, such as electro-olfactogram (EOG) or odor event–related potentials (OERPs) are used for research purposes only.

Olfactory threshold tests rely on measuring detection thresholds of a specific odorant, such as phenyl ethyl alcohol (PEA) or butyl alcohol. Odor identification tests are quantitative tests in which ~~patient~~**individual**s are asked to identify the odorants at the suprathreshold level. Examples include *The*

Connecticut odor identification, The University of Pennsylvania Identification Test (UPSIT) and the Cross-Cultural Smell Identification Test (CC-SIT). In Europe, a commonly used test is a threshold- and odorant-identification forced-choice test that uses odorant-impregnated felt-tipped pens (Sniffin' Sticks). A simple olfactory screening test using a 70% isopropyl alcohol pad as a stimulant has also been well described in the literature ~~(Wrobel, 2004)~~.[227]

Trigeminal Neuralgia (TN)~~:~~ – According to the International Headache Society, TN is defined as "a disorder characterized by recurrent unilateral brief electric shock-like pain, abrupt in onset and termination, limited to the distribution of one or more divisions of the trigeminal nerve and triggered by innocuous stimuli."[6] Atypical features include bilateral, hearing loss, dizziness/vertigo, visual changes, sensory loss, numbness, pain > 2min, pain outside trigeminal nerve distribution and progression.[114, 194]

Occipital Neuralgia~~:~~ – According to the International Headache Society, occipital neuralgia is defined "Unilateral or bilateral paroxysmal, shooting or stabbing pain in the posterior part of the scalp, in the distribution(s) of the greater, lesser and/or third occipital nerves, sometimes accompanied by diminished sensation or dysaesthesia in the affected area and commonly associated with tenderness over the involved nerve(s). Pain is eased temporarily by local anaesthetic block of the affected nerve(s). Occipital neuralgia must be distinguished from occipital referral of pain arising from the atlantoaxial or upper zygapophyseal joints or from tender trigger points in neck muscles or their insertions."[6]

MRI for Macrocephaly ~~–~~ Consider ultrasound in infants with macrocephaly and a normal neurological examination, no evidence of increased ICP and an open anterior fontanelle. If head US is normal, the infant should be monitored closely ~~(Smith, 1998)~~.[228] The anterior fontanelle generally closes between 10 and 24 months of age, with 3% closing between 5-9 months and 11% after 24 months ~~(Pindrik, 2014)~~.[229]

MRI and Normal Pressure Hydrocephalus (NPH) ~~–~~ Although diagnosis can be made based on CT findings alone, MRI is more accurate for disclosing associated pathologies (such as cerebrovascular disease), excluding other potential etiologies and for detecting NPH typical signs of prognostic value. A CT scan can exclude NPH and is appropriate for screening purposes and in ~~patient~~**individual**s who cannot undergo MRI ~~(Damasceno, 2015)~~.[141]

MRI and Vertigo – The most common causes of vertigo seen are benign paroxysmal positional vertigo (BPPV), vestibular neuronitis (VN) and Ménière's disease. These peripheral causes of vertigo are benign, and treatment involves reassurance and management of symptoms. Central causes of vertigo, such as cerebrovascular accidents (CVAs), tumors and multiple sclerosis (MS), need to be considered if the ~~patient~~**individual** presents with associated neurological symptoms, such as weakness, diplopia, sensory changes, ataxia, or confusion. Magnetic resonance imaging is appropriate in the evaluation of ~~patient~~**individual**s with vertigo who have neurologic signs and symptoms, progressive unilateral hearing loss or risk factors for cerebrovascular disease. MRI is more appropriate than CT for diagnosing vertigo due to its superiority in visualizing the posterior portion of the brain, where most central

nervous system disease that causes vertigo is found. A full neurologic and otologic evaluation including provocative maneuvers, vestibular function testing and audiogram can help evaluate vertigo of unclear etiology and differentiate between central and peripheral vertigo.

MRI and developmental delay – Significant ~~d~~~~evelopmental~~**developmental** delay is defined as significant delay (more than two standard deviations below the mean) in one or more developmental domains: gross/fine motor, speech/language, cognition, social/personal, and activities of daily living. Isolated delay in social/language development is characteristic of autism spectrum disorders or hearing loss. Isolated delay in motor development is characteristic of cerebral palsy (a static encephalopathy) or myopathy. Global developmental delay (GDD) is a subset of developmental delay defined as significant delay (by at least 2 SD's) in two or more developmental categories. Note that the term "GDD" is usually reserved for children <5 years old, whereas in older children >5 years, disability is quantifiable with IQ testing.
The yield of magnetic resonance imaging is low in children with autism spectrum disorder and no other neurologic findings; therefore, MRI is not recommended as a part of routine evaluation.[230]

Low risk brief resolved unexplained event (BRUE) formerly apparent life-threatening event (ALTE) requires all the following:

- Age > 60 days
- Gestational age ≥ 32 weeks or older and corrected gestational age ≥ 45 weeks
- First brief event
- Event lasting < 1 minute
- No CPR required by the trained medical provider
- No concerning historical features or physical examination findings.

Combination MRI/MRA of the Brain – This is one of the most misused combination studies and other than what is indicated above these examinations should be ordered in sequence, not together. Vascular abnormalities can be visualized on the brain MRI.

~~Patient~~**Individual**s presenting with a new migraine with aura (especially an atypical or complex aura) can mimic a transient ischemic attack or an acute stroke. If there is a new neurologic deficit, imaging should be guided by concern for cerebrovascular disease, not that the ~~patient~~**individual** has a headache ~~(Nahas, 2019)~~.[178, 231]

Leptomeningeal Carcinomatosis[232-235] ~~(Andersen, 2019; Clarke, 2010; Maillie, 2021; Wang, 2018)~~ – Leptomeningeal metastasis is an uncommon and typically late complication of cancer with poor prognosis and limited treatment options. Diagnosis is often challenging with nonspecific presenting symptoms ranging from headache and confusion to focal neurologic deficits such as cranial nerve palsies. Standard diagnostic evaluation involves a neurologic examination, MRI of the brain and spine with gadolinium, and cytologic evaluation of the cerebral spinal fluid (CSF). Hematologic malignancies (leukemia and lymphoma), primary brain tumors as well as solid malignancies can spread to the leptomeninges. The most common solid tumors giving rise to LM are breast cancer (12 - 35 %), small and non-small cell lung cancer (10-26 %), melanoma (5 -25 %), gastrointestinal malignancies (4-14 %), and cancers of unknown primary (1-7 %).

Drop Metastases – Drop metastases are intradural extramedullary spinal metastases that arise from intracranial lesions. Common examples of intracranial neoplasms that result in drop metastases include pineal tumors, ependymomas, medulloblastomas, germinomas, primitive neuroectodermal tumors (PNET), glioblastomas multiform, anaplastic astrocytomas, oligodendrogliomas and less commonly choroid plexus neoplasms and teratomas.[236]

POLICY HISTORY

Date	Summary
May 2022	Updated and reformatted references Updated background section Combo statements added Reorganized indications Changed visual deficits section added to background Reorganized suspected tumor section Clarified: • Acute headache, sudden onset • New onset headache related to activity or event (sexual activity, exertion, position), new or progressively worsening • Visual loss in background/removed note • Low flow vascular malformations • Histiocytic Neoplasms (Erdheim-Chester Disease, Langerhans Cell Histiocytosis, and Rosai-Dorfman Disease) for screening and/or with neurological signs or symptoms • Total testosterone levels persistently borderline around the lower limits of normal range (200-400 ng/dL) with low or normal LH/FSH; ○ *Low free testosterone* and consideration of reversible functional causes of gonadotropin suppression (e.g., obesity, opioid use, diabetes, steroid use or comorbid illness) • Follow-up of known CNS cancer (either primary malignant brain tumor or secondary brain metastasis) as per NCCN • Tumor monitoring in neurocutaneous syndromes as per tumor type • Histiocytic Neoplasms (Erdheim-Chester Disease, Langerhans Cell Histiocytosis, and Rosai-Dorfman Disease) To assess treatment response and surveillance of known brain lesions • To demonstrate dissemination in time for diagnosis (every 6-12 months) • To establish a new baseline (3-6 months after switching disease modifying therapy) • PML surveillance - Every 3-4 months, if high risk of PML occurrence; Brain MRI every 3–4 months for up to 12 months, in high-risk patients who switch from natalizumab to other therapeutics

	• Examples of mental status instruments to screen for cognitive impairment • For evaluation of new non-Parkinson neurological symptoms • Binocular diplopia with concern for intracranial pathology after comprehensive eye evaluation • Trigeminal neuralgia or *neuropathy*, notably with an atypical presentation • MRI Brain/MRI Orbit Combo – Optic Neuritis if atypical presentation (bilateral, absence of pain, optic nerve hemorrhages, severe visual impairment, lack of response to steroids, poor recovery, or recurrence • MRI Brain/MRI Face/Sinus/Neck Combo- Trigeminal neuralgia or neuropathy with an atypical presentation (for evaluation of the extracranial nerve course) **Added:** • Abnormal reflexes to neurologic deficit sections • 1-time screening for silent cerebral infarcts in school age children and adults with sickle cell disease • High stroke risk in sickle cell patients (2 - 16 years of age) with a transcranial doppler velocity > 200 • Midline dermoid cysts/sinuses with concern for intracranial extension • Elevated prolactin in the absence of other cause: > 100, persistently elevated or neuroendocrine signs or symptoms • Follow-up of known low grade tumor (WHO I-II) (i.e., meningioma, glioma, astrocytoma, oligodendroglioma) ○ For surveillance as per NCCN ○ If symptomatic, new/changing signs or symptoms or complicating factors • 6-month repeat scan in patients with MRI disease activity that is not associated with clinical activity on a follow-up scan (MS) • Note about pediatric MS imaging – same as adults except Increase frequency of imaging (e.g., every 6 months) in children with highly active disease or in situations where imaging will change management • Neurosarcoid ○ Initial Evaluation: ▪ Suspected based on neurological sign/symptoms and lab work (ACE, CSF analysis) OR ▪ Known history of sarcoidosis with neurological signs or symptoms ○ Follow up of known neurosarcoidosis:

	▪ To assess treatment response ▪ Worsening signs or symptoms • Tourette syndrome to list of movement disorders in which MRI is not indicated • Occipital Neuralgia • X-linked Adrenoleukodystrophy ○ Baseline MRI between 12 and 18 months old ○ Second MRI 1 year after baseline ○ MRI every 6 months between 3 and 12 years old ○ Annual MRI after 12 years old • Congenital/childhood sensorineural hearing loss suspected to be due to a structural abnormality (CNVIII, the brain parenchyma, or the membranous labyrinth). CT is the preferred imaging modality for the osseous anatomy and malformations of the inner. • Pulsatile tinnitus to combo section (MRI Brain with IAC/MRA Head/MRA Neck) • General Combo statement Note: These body regions might be evaluated separately or in combination as documented in the clinical notes by physical examination findings (e.g., localization to a particular segment of the neuroaxis), patient history, and other available information, including prior imaging. • Combo Brain MRI/MRA: ○ Neurological signs or symptoms in sickle cell patients ○ High stroke risk in sickle cell patients (2 - 16 years of age) with a transcranial doppler velocity > 200 • Brain MRI with IAC/ Brain MRA/Neck MRA (any combination) ○ Pulsatile tinnitus with concern for a suspected arterial vascular and/or intracranial etiology ○ Note: MRA and CTA are generally comparable noninvasive imaging alternatives each with their own advantages and disadvantages. Brain MRI can alternatively be combined with Brain CTA/Neck CTA. • MRI Brain/MRI Face/Sinus/Neck Combo- ○ Bell's Palsy/hemifacial spams for evaluation of the extracranial nerve course -if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset • MRI Brain/Spine Combo section ○ Drop metastasis from brain or spine

	○ **Combination studies for MS: These body regions might be evaluated separately or in combination as guided by physical examination findings (e.g., localization to a particular segment of the spinal cord), patient history (e.g., symptom(s), time course, and where in the CNS the likely localization(s) is/are), and other available information, including prior imaging** **Changed:** • **Thunderclap headache with continued concern for underlying vascular abnormality after initial negative brain imaging > 6 hours after onset (as well as in combo Brain MRI/MRA)** **Deleted:** • **Precocious puberty: and evidence of an accelerated bone age on x-y** • **Patient with history of CNS cancer (either primary or secondary) and a recent course of chemotherapy, radiation therapy (to the brain), or surgical treatment within the last two (2) years** • **Follow-up of known meningioma section/background**
~~May 2022~~	~~Updated and reformatted references~~ ~~Updated background section~~ ~~Combo statements added~~ ~~Reorganized indications~~ ~~Changed visual deficits section added to background~~ ~~Reorganized suspected tumor section~~ ~~Clarified:~~ - ~~Acute headache, sudden onset~~ - ~~New onset headache related to activity or event (sexual activity, exertion, position), new or progressively worsening~~ - ~~Visual loss in background/removed note~~ - ~~Low flow vascular malformations~~ - ~~Histiocytic Neoplasms (Erdheim-Chester Disease, Langerhans Cell Histiocytosis, and Rosai-Dorfman Disease) for screening and/or with neurological signs or symptoms~~ - ~~Total testosterone levels persistently borderline around the lower limits of normal range (200-400 ng/dL) with low or normal LH/FSH;~~ - ~~*Low free testosterone* and consideration of reversible functional causes of gonadotropin suppression (e.g., obesity, opioid use, diabetes, steroid use or comorbid illness)~~ - ~~Follow-up of known CNS cancer (either primary malignant brain tumor or secondary brain metastasis) as per ACCN~~ - ~~Tumor monitoring in neurocutaneous syndromes as per tumor type~~ - ~~Histiocytic Neoplasms (Erdheim-Chester Disease, Langerhans Cell Histiocytosis, and Rosai-Dorfman Disease) To assess treatment response and surveillance of known brain lesions~~

	- To demonstrate dissemination in time for diagnosis (every 6-12 months) - To establish a new baseline (3-6 months after switching disease modifying therapy) - PML surveillance - Every 3-4 months, if high risk of PML occurrence; Brain MRI every 3–4 months for up to 12 months, in high-risk patients who switch from natalizumab to other therapeutics - Examples of mental status instruments to screen for cognitive impairment - For evaluation of new non-Parkinson neurological symptoms - Binocular diplopia with concern for intracranial pathology after comprehensive eye evaluation - Trigeminal neuralgia or *neuropathy*, notably with an atypical presentation - **MRI Brain/MRI Orbit Combo** – Optic Neuritis if atypical presentation (bilateral, absence of pain, optic nerve hemorrhages, severe visual impairment, lack of response to steroids, poor recovery, or recurrence - **MRI Brain/MRI Face/Sinus/Neck Combo**- Trigeminal neuralgia or neuropathy with an atypical presentation (for evaluation of the extracranial nerve course) Added: - Abnormal reflexes to neurologic deficit sections - 1-time screening for silent cerebral infarcts in school age children and adults with sickle cell disease - High stroke risk in sickle cell patients (2 – 16 years of age) with a transcranial doppler velocity > 200 - Midline dermoid cysts/sinuses with concern for intracranial extension - Elevated prolactin in the absence of other cause: > 100, persistently elevated or neuroendocrine signs or symptoms - Follow-up of known low grade tumor (WHO I-II) (i.e., meningioma, glioma, astrocytoma, oligodendroglioma) - For surveillance as per ACCN - If symptomatic, new/changing signs or symptoms or complicating factors - 6-month repeat scan in patients with MRI disease activity that is not associated with clinical activity on a follow-up scan (MS) - Note about pediatric MS imaging – same as adults except Increase frequency of imaging (e.g., every 6 months) in children with highly active disease or in situations where imaging will change management - Neurosarcoid

	~~Initial Evaluation:~~ ~~Suspected based on neurological sign/symptoms and lab work (ACE, CSF analysis) OR~~ ~~Known history of sarcoidosis with neurological signs or symptoms~~ ~~Follow up of known neurosarcoidosis:~~ ~~To assess treatment response~~ ~~Worsening signs or symptoms~~ ~~Tourette syndrome to list of movement disorders in which MRI is not indicated~~ ~~Occipital Neuralgia~~ ~~X-linked Adrenoleukodystrophy~~ ~~Baseline MRI between 12 and 18 months old~~ ~~Second MRI 1 year after baseline~~ ~~MRI every 6 months between 3 and 12 years old~~ ~~Annual MRI after 12 years old~~ ~~Congenital/childhood sensorineural hearing loss suspected to be due to a structural abnormality (CNVIII, the brain parenchyma, or the membranous labyrinth). CT is the preferred imaging modality for the osseous anatomy and malformations of the inner.~~ ~~Pulsatile tinnitus to combo section (MRI Brain with IAC/MRA Head/MRA Neck)~~ ~~**General Combo statement**~~ ~~Note: These body regions might be evaluated separately or in combination as documented in the clinical notes by physical examination findings (e.g., localization to a particular segment of the neuroaxis), patient history, and other available information, including prior imaging.~~ ~~**Combo Brain MRI/MRA**:~~ ~~Neurological signs or symptoms in sickle cell patients~~ ~~High stroke risk in sickle cell patients (2 – 16 years of age) with a transcranial doppler velocity > 200~~ ~~**Brain MRI with IAC/ Brain MRA/Neck MRA (any combination)**~~ ~~Pulsatile tinnitus with concern for a suspected arterial vascular and/or intracranial etiology~~ ~~Note: MRA and CTA are generally comparable noninvasive imaging alternatives each with their own advantages and disadvantages. Brain MRI can alternatively be combined with Brain CTA/Neck CTA.~~ ~~**MRI Brain/MRI Face/Sinus/Neck Combo**- Bell's Palsy/hemifacial spams for evaluation of the extracranial nerve course -if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset~~

	~~MRI Brain/Spine Combo section~~ ~~Drop metastasis from brain or spine~~ ~~Combination studies for MS: These body regions might be evaluated separately or in combination as guided by physical examination findings (e.g., localization to a particular segment of the spinal cord), patient history (e.g., symptom(s), time course, and where in the CNS the likely localization(s) is/are), and other available information, including prior imaging~~ ~~Changed:~~ ~~Thunderclap headache with continued concern for underlying vascular abnormality after initial negative brain imaging > 6 hours after onset (as well as in combo Brain MRI/MRA)~~ ~~Deleted:~~ ~~Precocious puberty: and evidence of an accelerated bone age on x-y~~ ~~Patient with history of CNS cancer (either primary or secondary) and a recent course of chemotherapy, radiation therapy (to the brain), or surgical treatment within the last two (2) years~~ ~~Follow up of known meningioma section/background~~
November 2021	Added +0698T.
July 2021	**Reordered Indications** **Updated references** **Updated background section** **Added** • Brain MR/MRA are not approvable simultaneously unless they meet the criteria described below in the Indications for Brain MR/Brain MRA combination studies section. • Cluster headaches or other trigeminal-autonomic cephalgias i.e. paroxysmal hemicrania, hemicrania continua, short-lasting unilateral neuralgiform headache attacks (SUNCT/SUNA) imaging is indicated once to eliminate secondary causes (IHS, 2018) • Note: MRI is the study of choice for detecting cavernous malformations (CCM). Follow-up imaging of known CCM should be done only to guide treatment decisions or to investigate new symptoms. First-degree relatives of patients with more than one family member with a CCM should also have a screening MRI as well as genetic counseling • Langerhans cell histiocytosis with visual, neurological, or endocrine abnormality; polyuria or polydipsia; suspected craniofacial bone lesions, aural discharge, or suspected hearing impairment/mastoid involvement • Langerhans cell histiocytosis -To assess treatment response and surveillance of known brain lesions

	• Progressive Multifocal Leukoencephalopathy (PML) surveillance for patients on natalizumab (Tsyabri) o 12 months after the start of treatment in all patients o Further surveillance MRI scanning timing is based on anti-JCV antibody status ▪ If anti-JCV antibody negative, annually ▪ If anti-JCV antibody positive and antibody index < 1.5. every 6 months ▪ If anti-JCV antibody positive and antibody index > 1.5, every 3-4 months • Temporal Arteritis: Note: Protocol should include high-resolution contrast-enhanced imaging the temporal artery • similar mental status instruments */formal neuropsychological *Other examples include Ottawa 3DY (O3DY), Brief Alzheimer's Screen (BAS), Blessed Dementia Scale (BDS), caregiver-completed AD8 (cAD8), Brief Cognitive Rating Scale (BCRS), Clinical Dementia Rating (CDR) (Carptenter, 2011; McDougall, 1990) • FDA labeling for the drug Aduhelm (for Alzheimer's disease) requires baseline imaging and monitoring with Brain MRI. Criteria for coverage includes the following: o Baseline study within 1 year of initiating treatment unless the patient has a more recent exacerbation, traumatic event [e.g., falls, etc.], or co-morbidity necessitating an evaluation within one-month preceding initiation o Prior to the 7th and 12th infusions o Monitoring if radiographic severe Amyloid Related Imaging Abnormalities (ARIA) is suspected or observed NOTE: Enhanced clinical vigilance for ARIA is recommended during the first 8 doses of treatment with Aduhelm, particularly during titration. If a patient experiences symptoms which could be suggestive of ARIA, clinical evaluation should be performed, including MRI testing if indicated. • Optic atrophy as an abnormal eye finding • Childhood strabismus with development delay or abnormal fundoscopic exam to rule out intracranial abnormalities • Bulbar symptoms ie. difficulty in chewing, weakness of the facial muscles, dysarthria, palatal weakness, dysphagia, and dysphonia and/or signs i.e. atrophy and fasciculations of the tongue and absent gag reflex • Pseudobulbar symptoms i.e. dysphagia, dysarthria, facial weakness, sudden, stereotyped emotional outbursts that are not reflective of mood and/or signs i.e. spastic tongue and exaggerated gag/jaw jerk • Evaluation of the corticomedullary junction in Achondroplasia

	• Evaluation of suspected hydrocephalus with any acute, new, or fluctuating neurologic, motor, or mental status changes (separated this from known hydrocephalus) • Cisternography for intermittent and complex CSF rhinorrhea/otorrhea. CSF fluid should always be confirmed with laboratory testing (Beta-2 transferrin assay). • Suspected carotid or vertebral artery dissection with focal or lateralizing neurological deficits to Brain MRI/Brain MRA/Neck MRA combo • Headache associated with exercise or sexual activity (Brain MRI/Brain MRA combo) • Pre-operative evaluation for a planned surgery or procedure **Brain MRI/ Cervical MRI/Thoracic MRI (any combination)** o For evaluation of neuromyelitis optica spectrum disorders (recurrent or bilateral optic neuritis; recurrent transverse myelitis) o For known MS, prior to the initiation or change of disease modification treatments and assess disease burden (to establish a new baseline) o Follow -up scans for known MS if patients have known spine disease: ▪ 6-12 months after starting/changing treatment ▪ Every 1-2 years while on disease-modifying therapy to assess for subclinical disease activity, less frequently when stable for 2-3 years **Brain MRI/ Cervical MRI/Thoracic MRI/Lumbar (any combination)** • Follow up imaging of a known Arnold Chiari malformation (II/III). For Chiari, I follow-up imaging only if new or changing signs/symptoms o Suspected Leptomeningeal carcinomatosis (LC) o Tumor evaluation and monitoring in neurocutaneous syndromes - See Background o CSF leak highly suspected and supported by patient history and/or physical exam findings (known or suspected spontaneous (idiopathic) intracranial hypotension (SIH), post lumbar puncture headache, post spinal surgery headache, orthostatic headache, rhinorrhea or otorrhea, or cerebrospinal-venous fistula) **Brain MRI/Orbit MRI** Optic Neuritis- If needed to confirm optic neuritis and rule out compressive lesions **Clarified** • Symptoms indicative of increased intracranial pressure, such as recurring headaches after waking with or without associated nausea/vomiting

	• Suspected stroke with a personal or first-degree family history (brother, sister, parent, or child) of aneurysm or known coagulopathy or on anticoagulation • Symptoms of transient ischemic attack (TIA) (episodic neurologic symptoms such as sensory deficits, limb weakness, speech difficulties, visual loss, lack of coordination, or mental status changes) • Known or suspected skull fracture by physical exam and/or prior imaging • Neurologic findings (e.g. visual field deficit suggesting compression of the optic chiasm, diplopia, gaze palsy) – Pituitary • Follow-up known of pituitary adenoma - New neuroendocrine signs or symptoms • Follow of known arachnoid cyst (Al-Holou, 2010, 2013; Mustansir, 2018) ○ > 4 years old, repeat imaging only if newly symptomatic i.e. headaches, increased intracranial pressure, hydrocephalus, local mass effect, seizures, visual/endocrine dysfunction. • Temporal Arteritis - Atypical features, failure to response to treatment or concern for intracranial involvement • Central Nervous System (CNS) involvement in patients with known or suspected vasculitis or autoimmune disease with abnormal inflammatory markers or autoimmune antibodies • Suspected primary CNS vasculitis based on neurological signs and symptoms with completed infectious/inflammatory lab work-up • Anosmia or dysosmia on objective testing that is persistent and of unknown origin (also in combo section) • Trigeminal Neuralgia or other trigeminal autonomic cephalgias, notably in those with atypical presentation (also in combo section) • Clarified age < 18 for imaging of microcephaly and macrocephaly • For initial evaluation of a suspected Arnold Chiari malformation • For follow up imaging of a known Arnold Chiari malformation (II/III). For Chiari I follow-up imaging only if new or changing signs/symptoms • After full neurologic examination and vestibular testing with concern for central vertigo (i.e. skew deviation, vertical nystagmus, head thrust test, videonystagmography (VNG)/electronystagmography (ENG)) • Clarified age < 18 for imaging of developmental delay • Brain with IAC - CSF fluid should always be confirmed with laboratory testing (Beta-2 transferrin assay). • Optic neuropathy or unilateral optic disk swelling of unclear etiology (Brain MRI/Orbit MRI) **Deleted**

	• Approved indications as noted above and being performed in a child under 8 years of age who will need anesthesia for the procedure and there is a suspicion of concurrent vascular and intracranial pathology (redundant) • Brain MRI/Cervical MRI combo section (included in Brain MRI/ Cervical MRI/Thoracic MRI/Lumbar combos)
May 2020	Clarified: • New onset headache with (neurologic deficit) or with signs of increased intracranial pressure (papilledema) • Special additional considerations in the pediatric population with persistent headache ○ Documented absence of family history of headache • For evaluation of known or suspected stroke or vascular disease: • Suspected brain tumor • Suspected brain metastasis or intracranial involvement in patients with a history of cancer based on neurological symptoms or examination findings • Follow up of known malignant brain tumor Clarified: • Patient with history of CNS cancer (either primary or secondary) and a recent course of chemotherapy, radiation therapy (to the brain), or surgical treatment within the last two (2) years • Follow up of known non-malignant brain tumor/lesion if symptomatic, new/changing signs or symptoms or complicating factors • New onset of an unprovoked seizure in adults • Suspected intracranial abscess or brain infection • Suspected Encephalitis with headache and altered mental status or follow-up as clinically warranted • Mental status score of either MMSE or MoCA of less than 26 or other similar mental status instruments/neuropsychological testing Clarified: • Anosmia (loss of smell) documented by objective testing that is persistent and of unknown origin • Chiari malformation/syrinx Often congenital, but can present later in life; or less commonly acquired secondary to tumor, stroke, trauma, infection etc. • Vertigo associated with any of the following

	○ Risk factors for cerebrovascular disease with concern for stroke ○ After full neurologic examination and vestibular testing with concern for central vertigo • Combo Brain MRI/Orbit MRI ○ Reworded: Unilateral optic disk swelling/optic neuropathy of unclear etiology to distinguish between a compressive lesion of the optic nerve, optic neuritis, ischemic optic neuropathy (arteritic or non-arteritic), central retinal vein occlusion or optic nerve infiltrative disorders ○ Bilateral optic disk swelling (papilledema) with vision loss Added: • Visual loss (as a neurological deficit) Not explained by underlying ocular diagnosis, glaucoma or macular degeneration • Under New acute headache, sudden onset: ○ With a personal or family history of brain aneurysm or AVM (arteriovenous malformation) ○ Known coagulopathy or on anticoagulation • Under New onset of headache and any of the following ○ Fever ○ Subacute head trauma ○ Pregnancy or puerperium ○ Age ≥ 50 ○ Neurological deficits - Note: Neuroimaging warranted for atypical/complex migraine aura, but not for a typical migraine aura (see background) Added: • Special additional considerations in the pediatric population with persistent headache ○ Symptoms indicative of intracranial pressure, such as recurring headaches after waking with or without associated nausea/vomiting ○ Severe headache in a child with an underlying disease that predisposes to intracranial pathology (e.g.; immune deficiency, sickle cell disease neurofibromatosis, history of neoplasm, coagulopathy, hypertension, congenital heart disease) • Suspected stroke with a personal or family history (brother, sister, parent or child) of aneurysm or known coagulopathy/anticoagulation Added: • Suspected Pituitary Tumors: ○ With the following:

	▪ Neurologic findings (e.g. visual field deficit suggesting compression of the optic chiasm) ▪ Suspected hypofunctioning pituitary gland based on hormonal testing e.g., hypo pituitarism, growth hormone deficiency, hypogonadotropic hypogonadism [i.e. low gonadotropins (FSH/LH) and sex hormones*] ▪ * severe secondary hypogonadism with total testosterone persistently < 150 and low or normal LH/FSH OR ▪ * testosterone levels below normal range with low or normal LH/FSH AND • neurological sign and symptoms OR • other pituitary hormonal abnormalities OR • consideration of reversible functional causes of gonadotropin suppression (e.g. obesity, opioid use, or comorbid illness) Added: • Suspected hyperfunctioning pituitary gland based on hormonal testing i.e., central hyperthyroidism (high TSH), Cushing disease (high ACTH), acromegaly/gigantism (high GH/IGF-1) or elevated prolactin (>250 ng/mL or persistently elevated in the absence of another cause eg. stress, pregnancy, hypothyroidism, medication) • Note: Galactorrhea without elevated prolactin, imaging is not indicated • Central Diabetes Insipidus (low ADH) • Precocious puberty in a child (male < 9; female < 8), with hormonal studies suggesting a central cause and evidence of an accelerated bone age on X-ray • Pituitary apoplexy with sudden onset of neurological and hormonal symptoms • Suspected recurrence with prior history of CNS cancer based on neurological symptoms or examination Added: • Follow up of known meningioma ○ If <2cm or heavily calcified at 2 years and 5 years ○ > 2cm annually for 3 years and then scans at 5 years and 10 years. ○ Multiple meningiomas, annually ○ After treatment (surgery or radiotherapy), post-operative if concern for residual tumor, every 6-12 months then annually for 3-5 years based on WHO Grade (see background) • Follow-up known of pituitary adenoma ○ New signs or symptoms

	○ Functioning adenoma - to assess response to treatment and 1-year follow-up after drug holiday Added: • Follow of known pineal cyst (≥ 5mm) if there are atypical features or symptoms (e.g., headaches, gaze paresis, ataxia, papilledema, nausea/vomiting) • Follow of known arachnoid cyst ○ < 4 years old, serial imaging is warranted ○ > 4 years old, repeat imaging is approvable if newly symptomatic i.e. headaches, increased intracranial pressure, hydrocephalus, local mass effect, seizures, visual/endocrine dysfunction • For screening for known Non-CNS Cancer ○ Default screening for ▪ Kidney cancer ▪ Lung cancer ▪ Merkel cell carcinoma Added: • Mucosal melanoma of the head and neck, especially of the oral cavity • Poorly differential neuroendocrine cancer (Large or Small cell/Unknown primary of neuroendocrine origin) • Screening with preconditions ○ AML…….Suspicion of leukemic meningitis ○ Cutaneous melanoma….Stage IIIC or higher ○ Testicular Cancer-Seminoma….. High risk ○ Gestational Trophoblastic Neoplasia…Pulmonary metastasis ○ Bladder cancer……High risk, i.e. small cell • All other cancer if CNS symptoms present Added: • For screening of Hereditary Cancer Syndromes ○ Li Fraumeni syndrome- Annually ○ Von Hippel Lindau – Every 2 years, starting at age of 8 years ○ Tuberous Sclerosis – Every 1-3 years, until the age of 25 years ○ MEN1 – Every 3-5 years, starting at the age of 5 years ○ NF-2- Brain IAC: Annually starting, from age of 10 years ○ Sturge Weber Syndrome: Once, after age 1 to rule out intracranial involvement after; in patients <1 year, only if symptomatic • Known seizure disorder without previous imaging Added: • Imaging indications for new onset seizures in the pediatric population ○ Abnormal neurological exam, especially a postictal focal deficit

	○ Significant developmental delay ○ Focal onset ○ EEG shows focal or suspected structural abnormalities ○ <1 year of age Note: Imaging is not indicated in simple febrile seizures • Suspected temporal arteritis in a patient > 50 with temporal headache, abrupt visual changes, jaw claudication, temporal artery tenderness, constitutional symptoms or elevated ESR AND ○ Negative initial work-up (color Doppler ultrasonography or biopsy) OR ○ Atypical features or failure to response to treatment with concern for large vessel involvement Added: • MRI indicted for atypical dystonia. Note: MRI not indicated in essential tremor or isolated focal dystonia (e.g., blepharospasm, cervical dystonia, laryngeal dystonia, oromandibular dystonia, writer's dystonia) • Binocular diplopia with concern for intracranial pathology • Hemifacial spasm • Other objective cranial nerve palsy (CN IX-XII) • Follow up shunt evaluation (Pople, 2002, Reddy, 2014, Kamenova, 2018) ○ Post operatively if indicated based on underlying disease and pre-operative radiographic findings and/or ○ 6-12 months after placement and/or ○ With neurologic symptoms that suggest shunt malfunction Added: • Suspected spontaneous intra-cranial hypotension with distinct postural headache other symptoms include: nausea, vomiting, dizziness, tinnitus, diplopia neck pain or imbalance • CSF flow study for evaluation and management of CSF flow disorders • Diagnosis of central sleep apnea on polysomnogram ○ Children > 1 year ○ Adults in the absence of heart failure, chronic opioid use, high altitude, or treatment emergent central sleep apnea AND concern for a central neurological cause (Chiari malformation, tumor, infectious/inflammatory disease) OR with an abnormal neurological exam • Syncope with clinical concern for seizure or associated neurological signs or symptoms • Cyclical vomiting syndrome or abdominal migraine with any localizing neurological symptoms

	• Soft tissue mass of the head with nondiagnostic initial evaluation (ultrasound and/or radiograph) Added: • Cerebral palsy if etiology has not been established the neonatal period, there is change in the expected clinical or developmental profile or concern for progressive neurological disorder • Unexplained event (BRUE) formerly apparent life-threatening event (ALTE) in infants < 1 year with concern for neurological cause based on history and exam Note: Imaging is not indicated in low risk patients • Under Indications for a Brain MRI with Internal Auditory Canal (IAC): ○ CSF otorrhea (MRI for intermittent leak, CT for active leaks) ○ Clinical Suspicion of acute mastoiditis as a complication of acute otitis media with intracranial complications (i.e. meningeal signs, cranial nerve deficits, focal neurological findings, altered mental status) ○ Bell's Palsy for evaluation of the extracranial nerve course - if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset Added: • Combo Brain MRI/MRA ○ Thunderclap headache with continued concern for underlying vascular abnormality after initial negative work-up ▪ Negative Brain CT; ▪ AND Negative Lumbar Puncture ▪ Acute, sudden onset of headache with personal history of a vascular abnormality or first-degree family history of aneurysm • Combo Brain MRI/Orbit MRI ○ Optic Neuritis if atypical presentation, severe visual impairment or poor recovery following initial onset or treatment onset • Combo Brain MRI/Face/Sinus/Neck MRI ○ Bells/hemifacial spasm that meets above criteria ○ Objective cranial nerve palsy (CN IX-XII) (for evaluation of the extracranial nerve course ○ Granulomatosis with polyangiitis (Wegener's granulomatosis) disease Deleted: • Under New onset of headache and any of the following

	○ Temporal headache in person > 55, with sedimentation rate (ESR) > 55 with tenderness over the temporal artery. • Known or suspected pituitary tumor with corroborating physical exam (i.e., galactorrhea or acromegaly) neurologic findings and/or lab abnormalities. • Known brain tumor and new onset of headache. • Follow up shunt evaluation within six (6) months of placement or one (1) year follow up and/or with neurologic symptoms • From combo Brain MRI/MRA Clinical suspicion of subarachnoid hemorrhage (SAH) ie thunderclap headache
August 2019	• For evaluation of patient with neurologic symptoms or deficits suspicious for MS: Added: "clinically isolated syndrome OR recurrent episodes of variable neurological signs or symptoms not attributable to another cause; And Removed time frame of 'within the last 4 weeks' • Removed: Stable condition with no prior imaging within the past ten (10) months or within the past six (6) months if patient has relapsing disease • Removed: Exacerbation of symptoms or change in symptom characteristics such as frequency or type and demonstrated compliance with medical therapy. • For evaluation of MS, added: ○ To establish a new baseline (no recent imaging, postpartum, or 6-12 months after switching disease modifying therapy) ○ Prior to starting or switching disease-modifying therapy ○ Every 1-2 years while on disease-modifying therapy to assess for subclinical disease activity, less frequently when stable for 2-3 years ○ New signs or symptoms suggested of an exacerbation or unexpected clinical worsening ○ PML surveillance for patients on natalizumab • For evaluation of known or suspected seizure disorder, added: ○ Newly identified change in seizure activity/pattern • Renamed Parkinson's section to: Movement disorders and added: ○ For the evaluation of other movement disorder to exclude a structural lesion (i.e., suspected Huntington disease, chorea, atypical parkinsonian syndromes, hemiballismus, secondary dystonia). ○ * MRI not indicated in essential tremor or primary dystonia ○ For suspected Parkinson's, added 'with atypical feature or unresponsive to levodopa

	• For evaluation of neurologic symptoms or deficits, added: visual loss
	• For trauma, added:
	  ○ On anticoagulation
	  ○ Post concussive syndrome if persistent or disabling symptoms and imaging has not been performed
	  ○ Subacute or chronic traumatic brain injury with new cognitive and/or neurologic deficit
	• For evaluation of headache, added or removed:
	  ○ Prior history of stroke or intracranial bleed with sudden onset of severe headache (moved)
	  ○ New headache and signs of increased intracranial pressure
	  ○ Related to activity or event (sexual activity, exertion, position) (new or progressively worsening)
	  ○ New headache and persistent or progressively worsening during a course of physician directed treatment
	  ○ Special considerations in the pediatric population with persistent headache:
	  ▪ Occipital location
	  ▪ Age < 6 years
	  ▪ No family history of headache
	• For evaluation of brain tumor:
	  ○ Specified 'malignant' for f/u of known tumor
	  ○ Added: Follow up of known benign tumor if symptomatic, new/changing signs or symptoms or complicating factors; Follow up of known meningioma; and tumor evaluation and monitoring in neurocutaneous syndromes
	  ○ Removed: Known lung cancer or rule out metastasis and/or preoperative evaluation, Metastatic melanoma (not all melanomas)
	• For evaluation of suspected stroke:
	  ○ Moved 'patient with history of a known stroke with new and sudden onset of severe headache'
	  ○ Separated: Family history of aneurysm
	• For evaluation inflammatory disease or infections:
	  ○ Changed meningitis with positive signs and symptoms from 'And' positive lab findings to 'OR' positive labs
	  ○ For suspected encephalitis removed 'severe' headache
	• For evaluation of congenital abnormality:
	  ○ Modified the age restriction of > 6 months age for eval of macrocephaly to include 'in an infant/child with previously

	abnormal US, abnormal neurodevelopmental exam, signs of increased ICP or closed anterior fontanelle' • For known or suspected normal pressure hydrocephalus (NPH): o Added - With symptoms of gait difficulty, cognitive disturbance and urinary incontinence • Other Indications: o Added detail to Vertigo including: ▪ Signs or symptoms suggestive of a CNS lesion (ataxia, visual loss, double vision, weakness or a change in sensation) ▪ Progressive unilateral hearing loss ▪ Risk factors for cerebrovascular disease ▪ After full neurologic examination and ENT work-up with concern for central vertigo o Modified developmental delay to include: Global developmental delay or developmental delay with abnormal neurological examination o Added: ▪ Horner's syndrome with symptoms localizing the lesion to the central nervous system ▪ Trigeminal Neuralgia – if <40 years of age or atypical features (ie bilateral, hearing loss, dizziness/vertigo, visual changes, sensory loss, numbness, pain >2min, pain outside trigeminal nerve distribution, progression) ▪ Bell's Palsy- if atypical signs, slow resolution beyond three weeks, no improvement at four months, or facial twitching/spasms prior to onset. ▪ Psychological changes with neurological deficits on exam or after completion of a full neurological assessment that suggests a possible neurologic cause ▪ New onset anisocoria o Removed Objective cranial nerve palsy; and Cholesteatoma (duplicated) • For Brain MRI/Neck MRA: deleted 'confirmed carotid occlusion > 60%, surgery or angioplasty candidate' and added 'Suspected carotid or vertebral artery dissection with focal or lateralizing neurological deficits' • Added Brain MRI/Brain MRA section, including: Clinical suspicion of subarachnoid hemorrhage (SAH) ie thunderclap headache; and Suspected venous thrombosis (dural sinus thrombosis) • Added Brain MRI/Brain MRA/Neck MRA section, including: Recent stroke or transient ischemic attack (TIA); and Approved indications as noted above and being performed in a child under 8 years of age who will need

	anesthesia for the procedure and there is a suspicion of concurrent vascular and intracranial pathology • For Brain MRI/Cervical MRI, added: Suspected MS with new or changing symptoms consistent with cervical spinal cord disease; and Follow up to the initiation or change in medication for patient with known Multiple Sclerosis • For Brain MRI/Orbit MRI, added: Bilateral papilledema with visual loss; and Known or suspected neuromyelitis optica spectrum disorder with severe, recurrent or bilateral optic neuritis; AND changed age restriction from 3 years to 8 years for children requiring anesthesia for the procedure with suspicion of concurrent orbital and intracranial pathology or tumor • Added section for Brain MRI/Face/Sinus/Neck MRI, including: Anosmia on objective testing; and Trigeminal neuralgia or cranial nerve palsy that meets the above criteria • Updated background information and references

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~~**Reviewed / Approved by NIA Clinical Guideline Committee**~~

~~GENERAL INFORMATION~~

~~It is an expectation that all patients receive care/services from a licensed clinician. All appropriate supporting documentation, including recent pertinent office visit notes, laboratory data, and results of any special testing must be provided. If applicable: All prior relevant imaging results and the reason that alternative imaging cannot be performed must be included in the documentation submitted.~~

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ADDITIONAL RESOURCES

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Reviewed / Approved by NIA Clinical Guideline Committee

GENERAL INFORMATION

It is an expectation that all patients receive care/services from a licensed clinician. All appropriate supporting documentation, including recent pertinent office visit notes, laboratory data, and results of any special testing must be provided. If applicable: All prior relevant imaging results and the reason that alternative imaging cannot be performed must be included in the documentation submitted.